

THE CENTURY PSYCHOLOGY SERIES

RICHARD M. ELLIOTT, *Editor*

Principles of Behavior

Principles of Behavior

AN INTRODUCTION TO BEHAVIOR THEORY

by

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PREFACE

As suggested by the title, this book attempts to present in an objective, systematic manner the primary, or fundamental, molar principles of behavior. It has been written on the assumption that all behavior, individual and social, moral and immoral, normal and psychopathic, is generated from the same primary laws; that the differences in the objective behavioral manifestations are due to the differing conditions under which habits are set up and function. Consequently the present work may be regarded as a general introduction to the theory of all the behavioral (social) sciences.

In an effort to insure its intelligibility to all educated readers, the complicated equations and other more technical considerations have been relegated to terminal notes, where they may be found by the technically trained who care to consult them. The formal verbal statements of the primary principles are presented in special type at the ends of the chapters in which they emerge from the analysis. A convenient glossary of the various symbols employed is provided.

There remains the pleasant duty of recording my numerous obligations. First in order of importance is my gratitude to the Institute of Human Relations and to its Director, Mark A. May, for the leisure and the generous provision of innumerable accessory facilities which have made possible the preparation of this work, and for the stimulation and instruction received during several years of Monday night Institute staff meetings. Contributing to the same end have been the stimulation, criticism, and suggestions given by the members of my seminar in psychology in the Yale Graduate School. Few things have been so pleasant and so profitable scientifically as the contacts with the brilliant and vigorous young personalities encountered in these situations. The most of whatever is novel in the pages of this book has been generated in one way or another from these contacts.

To certain individuals a more specific debt of gratitude must be acknowledged. All the original figures were drawn by Joy Richardson and Don Olson. Bengt Carlson fitted equations to the

numerous empirical curves, and is responsible for all of the more technical mathematical material which appears in the terminal notes. Marvin J. Herbert prepared the subject index. To my laboratory research assistants of the last ten years, Walter C. Shipley, St. Clair A. Switzer, Milton J. Bass, Eliot H. Rodnick, Carl Iver Hovland, Douglas G. Ellson, Richard Bugelski, Glen L. Heathers, Peter Arakelian, Chester J. Hill, John L. Finan, Stanley B. Williams, C. Theodore Perin, Richard O. Rouse, Charles B. Woodbury, and Ruth Hays, I am indebted for the conscientious performance of numerous experiments which were especially planned for this work, and which naturally make up a considerable proportion of the empirical material employed. The loyal coöperation and kindly day by day suggestions and criticisms of these splendid young people have made that phase of the task a rarely satisfying labor.

Another and smaller group of individuals have given invaluable aid in the preparation of the manuscript. Eleanor Jack Gibson read much of an early version of the manuscript and made helpful suggestions. Irvin L. Child read the manuscript in a late stage of revision and made numerous valuable criticisms and suggestions. To Kenneth L. Spence I owe a debt of gratitude which cannot adequately be indicated in this place; from the time when the ideas here put forward were in the process of incubation in my graduate seminar and later when the present work was being planned, on through its many revisions, Dr. Spence has contributed generously and effectively with suggestions and criticisms, large numbers of which have been utilized without indication of their origin. Finally, to Ruth Hays I am deeply indebted for the transcription of hundreds of pages of unbelievably illegible handwriting, for the preparation of the name index, and for absolutely indispensable assistance with the formal aspects of the manuscript.

C. L. H.

New Haven

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Principles of Behavior

CHAPTER I

The Nature of Scientific Theory

This book is the beginning of an attempt to sketch a systematic objective theory of the behavior of higher organisms. It is accordingly important at the outset to secure a clear notion of the essential nature of systematic theory in science, the relation of theory to other scientific activities, and its general scientific status and importance.

THE TWO ASPECTS OF SCIENCE: EMPIRICAL AND EXPLANATORY

Men are ever engaged in the dual activity of making observations and then seeking explanations of the resulting revelations. All normal men in all times have observed the rising and setting of the sun and the several phases of the moon. The more thoughtful among them have then proceeded to ask the question, "Why? Why does the moon wax and wane? Why does the sun rise and set, and where does it go when it sets?" Here we have the two essential elements of modern science: the making of observations constitutes the empirical or factual component, and the systematic attempt to explain these facts constitutes the theoretical component. As science has developed, specialization, or division of labor, has occurred; some men have devoted their time mainly to the making of observations, while a smaller number have occupied themselves largely with the problems of explanation.

During the infancy of science, observations are for the most part casual and qualitative—the sun rises, beats down strongly at midday, and sets; the moon grows from the crescent to full and then diminishes. Later observations, usually motivated by practical considerations of one kind or another, tend to become quantitative and precise—the number of days in the moon's monthly cycle are counted accurately, and the duration of the sun's yearly course is determined with precision. As the need for more exact observations increases, special tools and instruments, such as graduated measuring sticks, protractors, clocks, telescopes, and microscopes, are devised to facilitate the labor. Kindred tools relating to a given field of science are frequently assembled under a single

roof for convenience of use; such an assemblage becomes a laboratory.

As scientific investigations become more and more searching it is discovered that the spontaneous happenings of nature are not adequate to permit the necessary observations. This leads to the setting up of special conditions which will bring about the desired events under circumstances favorable for such observations; thus experiments originate. But even in deliberate experiment it is often extraordinarily difficult to determine with which among a complex of antecedent conditions a given consequence is primarily associated; in this way arise a complex maze of control experiments and other technical procedures, the general principles of which are common to all sciences but the details of which are peculiar to each. Thus in brief review we see the characteristic technical development of the empirical or factual aspect of science.

Complex and difficult as are some of the problems of empirical science, those of scientific theory are perhaps even more difficult of solution and are subject to a greater hazard of error. It is not a matter of chance that the waxing and waning of the moon was observed for countless millennia before the comparatively recent times when it was at last successfully explained on the basis of the Copernican hypothesis. Closely paralleling the development of the technical aids employed by empirical science, there have also grown up in the field of scientific theory a complex array of tools and special procedures, mostly mathematical and logical in nature, designed to aid in coping with these peculiar difficulties. Because of the elementary nature of the present treatise, very little explicit discussion of the use of such tools will be given.

THE DEDUCTIVE NATURE OF SCIENTIFIC THEORY AND EXPLANATION

The term *theory* in the behavioral or "social" sciences has a variety of current meanings. As understood in the present work, a theory is a systematic deductive derivation of the secondary principles of observable phenomena from a relatively small number of primary principles or postulates, much as the secondary principles or theorems of geometry are all ultimately derived as a logical hierarchy from a few original definitions and primary principles called axioms. In science an observed event is said to be explained when the proposition expressing it has been logically derived from

a set of definitions and postulates coupled with certain observed conditions antecedent to the event. This, in brief, is the nature of scientific theory and explanation as generally understood and accepted in the physical sciences after centuries of successful development (1, pp. 495-496).

The preceding summary statement of the nature of scientific theory and explanation needs considerable elaboration and exemplification. Unfortunately the finding of generally intelligible examples presents serious difficulties; because of the extreme youth of systematic behavior theory (1, p. 501 ff.; 2, p. 15 ff.) as here understood, it is impossible safely to assume that the reader possesses any considerable familiarity with it. For this reason it will be necessary to choose all the examples from such physical sciences as are now commonly taught in the schools.

We can best begin the detailed consideration of the nature of scientific explanation by distinguishing it from something often confused with it. Suppose a naïve person with a moderate-sized telescope has observed Venus, Mars, Jupiter, and Saturn, together with numerous moons (including our own), and found them all to be round in contour and presumably spherical in form. He might proceed to formulate his observations in a statement such as, "All heavenly bodies are spherical," even though this statement goes far beyond the observations, since he has examined only a small sample of these bodies. Suppose, next, he secures a better telescope; he is now able to observe Uranus and Neptune, and finds both round in contour also. He may, in a manner of speaking, be said to explain the sphericity of Neptune by subsuming it under the category of heavenly bodies and then applying his previous empirical generalization. Indeed, he could have predicted the spherical nature of Neptune by this procedure before it was observed at all:

All heavenly bodies are spherical.
Neptune is a heavenly body,
Therefore Neptune is spherical.

Much of what is loosely called explanation in the field of behavior is of this nature. The fighting propensities of a chicken are explained by the fact that he is a game cock and game cocks are empirically known to be pugnacious. The gregariousness of a group of animals is explained by the fact that the animals in question are dogs, and dogs are empirically known to be gregarious.

As we have seen, it is possible to make concrete predictions of a sort on the basis of such generalizations, and so they have significance. Nevertheless this kind of procedure—the subsumption of a particular set of conditions under a category involved in a previously made empirical generalization—is not exactly what is regarded here as a scientific theoretical explanation.

For one thing, a theoretical explanation as here understood grows out of a problem, e.g., "What must be the shape of the heavenly bodies?" Secondly, it sets out from certain propositions or statements. These propositions are of two rather different kinds. Propositions of the first type required by an explanation are those stating the relevant initial or *antecedent conditions*. For example, an explanation of the shape of heavenly bodies might require the preliminary assumption of the existence of (1) a large mass of (2) more or less plastic, (3) more or less homogeneous matter, (4) initially of any shape at all, (5) the whole located in otherwise empty space. But a statement of the antecedent conditions is not enough; there must also be available a set of statements of *general principles* or rules of action relevant to the situation. Moreover, the particular principles to be utilized in a given explanation must be chosen from the set of principles generally employed by the theorist in explanations of this class of phenomena, the choice to be made strictly on the basis of the nature of the question or problem under consideration taken in conjunction with the observed or assumed conditions. For example, in the case of the shape of the heavenly bodies the chief principle employed is the Newtonian law of gravitation, namely, that every particle of matter attracts every other particle to a degree proportional to the product of their masses and inversely proportional to the square of the distances separating them. These principles are apt themselves to be verbal formulations of empirical generalizations, but may be merely happy conjectures or guesses found by a certain amount of antecedent trial-and-error to agree with observed fact. At all events they originate in one way or another in empirical observation.

The concluding phase of a scientific explanation is the derivation of the answer to the motivating question from the conditions and the principles, taken jointly, by a process of inference or reasoning. For example, it follows from the principle of gravitation that empty spaces which might at any time have existed within the mass of a heavenly body would at once be closed. Moreover,

if at any point on the surface there were an elevation and adjacent to it a depression or valley, the sum of the gravitational pressures of the particles of matter in the elevation acting on the plastic material beneath would exert substantially the same pressure laterally as toward the center of gravity. But since there would be no equal lateral pressure originating in the valley to oppose the pressure originating in the elevation, the matter contained in the elevation would flow into the valley, thus eliminating both. This means that in the course of time all the matter in the mass under consideration would be arranged about its center of gravity with no elevations or depressions; i.e., the radius of the body at all points would be the same. In other words, if the assumed mass were not already spherical it would in the course of time automatically become so (4, p. 424). It follows that all heavenly bodies, including Neptune, must be spherical in form.

The significance of the existence of these two methods of arriving at a verbal formulation of the shape of the planet Neptune may now be stated. The critical characteristic of scientific theoretical explanation is that it reaches independently through a process of reasoning the same outcome with respect to (secondary) principles as is attained through the process of empirical generalization. Thus scientific theory may arrive at the general proposition, "All heavenly bodies of sufficient size, density, plasticity, and homogeneity are spherical," as a theorem, simply by means of a process of inference or deduction without any moons or planets having been observed at all. The fact that, in certain fields at least, practically the same statements or propositions can be attained quite independently by empirical methods as by theoretical procedures is of enormous importance for the development of science. For one thing, it makes possible the checking of results obtained by one method against those obtained by the other. It is a general assumption in scientific methodology that if everything entering into both procedures is correct, the statements yielded by them will never be in genuine conflict.

SCIENTIFIC EXPLANATIONS TEND TO COME IN CLUSTERS CONSTITUTING A LOGICAL HIERARCHY

This brings us to the important question of what happens in a theoretical situation when one or more of the supposed antecedent conditions are changed, even a little. For example, when

considering the theoretical shape of heavenly bodies, instead of the mass being completely fluid it might be assumed to be only slightly plastic. It is evident at once, depending on the degree of plasticity, the size of the mass, etc., that there may be considerable deviation from perfect sphericity, such as the irregularities observable on the surface of our own planet. Or suppose that we introduce the additional condition that the planet revolves on its axis. This necessarily implies the entrance into the situation of the principle of centrifugal force, the familiar fact that any heavy object whirled around in a circle will pull outward. From this, in conjunction with other principles, it may be reasoned (and Newton did so reason) that the otherwise spherical body would bulge at the equator; moreover, this bulging at the equator together with the principle of gravity would, in turn, cause a flattening at the poles (4, p. 424). Thus we see how it is that as antecedent conditions are varied the theoretical outcome (theorem) following from these conditions will also vary. By progressively varying the antecedent conditions in this way an indefinitely large number of theorems may be derived, but all from the very same group of basic principles. The principles are employed over and over in different combinations, one combination for each theorem. Any given principle may accordingly be employed many times, each time in a different context. In this way it comes about that scientific theoretical systems potentially have a very large number of theorems (secondary principles) but relatively few general (primary) principles.

We note, next, that in scientific systems there are not only many theorems derived by a process of reasoning from the same assemblage of general principles, but these theorems take the form of a logical hierarchy: first-order theorems are derived directly from the original general principles; second-order theorems are derived with the aid of the first-order theorems; and so on in ascending hierarchical orders. Thus in deducing the flattening of the planets at the poles, Newton employed the logically antecedent principle of centrifugal force which, while an easily observable phenomenon, can itself be deduced, and so was deduced by Newton, from the conditions of circular motion. The principle of centrifugal force accordingly is an example of a lower-order theorem in Newton's theoretical system (4, p. 40 ff.). On the other hand, Newton derived from the bulging of the earth at its equator what is known as the "precession of the equinoxes" (4, p. 580), the fact that the length of the year as determined by the time elapsing from one occasion

when the shadow cast by the winter sun at noon is longest to the next such occasion, is shorter by some twenty minutes than the length of the year as determined by noting the time elapsing from the conjunction of the rising of the sun with a given constellation of stars to the next such conjunction. This striking phenomenon, discovered by Hipparchus in the second century B.C., was first explained by Newton. The precession of the equinoxes accordingly is an example of a higher-order theorem in the Newtonian theoretical system.

From the foregoing it is evident that in its deductive nature systematic scientific theory closely resembles mathematics. In this connection the reader may profitably recall his study of geometry with (1) its definitions, e.g., point, line, surface, etc., (2) its primary principles (axioms), e.g., that but one straight line can be drawn between two points, etc., and following these (3) the ingenious and meticulous step-by-step development of the proof of one theorem after the other, the later theorems depending on the earlier ones in a magnificent and ever-mounting hierarchy of derived propositions. Proper scientific theoretical systems conform exactly to all three of these characteristics.¹ For example, Isaac Newton's *Principia* (4), the classical scientific theoretical system of the past, sets out with (1) seven definitions concerned with such notions as matter, motion, etc., and (2) a set of postulates consisting of his three famous laws of motion, from which is derived (3) a hierarchy of seventy-three formally proved theorems together with large numbers of appended corollaries. The theorems and corollaries are concerned with such concrete observable phenomena as centrifugal force, the shape of the planets, the precession of the equinoxes, the orbits of the planets, the flowing of the tides, and so on.

SCIENTIFIC THEORY IS NOT ARGUMENTATION

The essential characteristics of scientific theory may be further clarified by contrasting it with argumentation and even with geometry. It is true that scientific theory and argument have similar formal or deductive structures; when ideally complete both should have their terms defined, their primary principles stated,

¹The formal structure of scientific theory differs in certain respects from that of pure mathematics, but these differences need not be elaborated here; the point to be emphasized is that mathematics and scientific theory are alike in that they are both strictly deductive in their natures.

and their conclusions derived in an explicit and logical manner. In spite of this superficial similarity, however, the two differ radically in their essential natures, and it would be difficult to make a more serious mistake than to confuse them. Because of the widespread tendency to just this confusion, the distinction must be stressed. An important clue to the understanding of the critical differences involved is found in the objectives of the two processes.

The primary objective of argumentation is persuasion. It is socially aggressive; one person is deliberately seeking to influence or coerce another by means of a process of reasoning. There is thus in argumentation a proponent and a recipient. On the surface the proponent's objective often appears to be nothing more than to induce the recipient to assent to some more or less abstract proposition. Underneath, however, the ultimate objective is usually to lead the recipient to some kind of action, not infrequently such as to be of advantage to the proponent or some group with which the proponent is allied. Now, for the effort involved in elaborate argumentation to have any point, the proposition representing the objective of the proponent's efforts must be of such a nature that it cannot be substantiated by direct observation. The recipient cannot have made such observations; otherwise he would not need to be convinced.

Moreover, for an argument to have any coerciveness, the recipient must believe that the definitions and the other basic assumptions of the argument are sound; the whole procedure is that of systematically transferring to the final culminating conclusion the assent which the recipient initially gives to these antecedent statements. In this connection it is to be noted that systems of philosophy, metaphysics, theology, etc., are in the above sense at bottom elaborate arguments or attempts at persuasion, since their conclusions are of such a nature that they cannot possibly be established by direct observation. Consider, for example, Proposition XIV of Part One of Spinoza's *Ethic* (5):

"Besides God no substance can be, . . ."

The primary objective of scientific theory, on the other hand, is the establishment of scientific principles. Whereas argumentation is socially aggressive and is directed at some other person, natural science theory is aggressive towards the problems of nature, and it uses logic as a tool primarily for mediating to the scientist himself a more perfect understanding of natural processes. If New-

ton had been a scientific Robinson Crusoe, forever cut off from social contacts, he would have needed to go through exactly the same logical processes as he did, if he were *himself* to have understood why the heavenly bodies are spherical rather than cubical. Naturally also, argumentation presupposes that the proponent has the solution of the question at issue fully in hand; hence his frequent overconfidence, aggressiveness, and dogmatism. In contrast to this, the theoretical activities of science, no less than its empirical activities, are directed modestly toward the gradual, piecemeal, successive-approximation establishment of scientific truths. In a word, scientific theory is a technique of investigation, of seeking from nature the answers to questions motivating the investigator; it is only incidentally and secondarily a technique of persuasion. It should never descend to the level of mere verbal fencing so characteristic of metaphysical controversy and argumentation.

Some forms of argumentation, such as philosophical and metaphysical speculation, have often been supposed to attain certainty of their conclusions because of the "self-evident" nature of their primary or basic principles. This is probably due to the influence of Euclid, who believed his axioms to be "self-evident truths." At the present time mathematicians and logicians have largely abandoned intuition or self-evidentiality as a criterion of basic or any other kind of truth. Similarly, scientific theory recognizes no axiomatic or self-evident truths; it has postulates but no axioms in the Euclidian sense. Not only this; scientific theory differs sharply from argumentation in that its postulates are not necessarily supposed to be true at all. In fact, scientific theory largely inverts the procedure found in argument: *whereas argument reaches belief in its theorems because of antecedent belief in its postulates, scientific theory reaches belief in its postulates to a considerable extent through direct or observational evidence of the soundness of its theorems* (2, p. 7).

THEORETICAL AND EMPIRICAL PROCEDURES CONTRIBUTE JOINTLY TO THE SAME SCIENTIFIC END

No doubt the statement that scientific theory attains belief in its postulates through belief in the soundness of its theorems will come as a distinct surprise to many persons, and for several reasons. For one thing, the thoughtful individual may wonder why, in spite

of the admitted absence of self-evident principles or axioms, the basic principles of scientific systems are not firmly established at the outset by means of observation and experiment. After such establishment, it might be supposed, the remaining theorems of the system could all be derived by an easy logical procedure without the laborious empirical checking of each, as is the scientific practice. Despite the seductive charm of its simplicity this methodology is, alas, impossible. One reason is, as already pointed out (p. 3), that the generalizations made from empirical investigations can never be quite certain. Thus, as regards the purely empirical process every heavenly body so far observed might be spherical, yet this fact would only increase the probability that the next one encountered would be spherical; it would not make it certain. The situation is exactly analogous to that of the continued drawing of marbles at random from an urn containing white marbles and suspected of containing black ones also. As one white marble after another is drawn in an unbroken succession the probability increases that the next one drawn will be white, but there can never come a time when there will not be a margin of uncertainty. *On the basis of observation alone* to say that all heavenly bodies are spherical is as unwarranted as it would be to state positively that all the marbles in an urn must be white because a limited random sampling has been found uniformly to be white.

But even for the sampling of empirical theory or experimental truth to be effective, the sampling of the different situations involved must be truly random. This means that the generalization in question must be tried out empirically with all kinds of antecedent conditions; which implies that it must be tested in conjunction with the operation of the greatest variety of other principles, singly and in their various combinations. In *very* simple situations the scientist in search of primary principles needs to do little more than formulate his observations. For example, it is simple enough to observe the falling of stones and similar heavy objects, and even to note that such objects descend more rapidly the longer the time elapsed since they were released from rest. But the moment two or more major principles are active in the same situation, the task of determining the rôle played by the one under investigation becomes far more difficult. It is not at all obvious to ordinary observation that the principle of gravity operative in the behavior of freely falling bodies is the same as that operative in the behavior

of the common pendulum; ordinary falling bodies, for example, do not manifest the phenomena of lateral oscillation. The relation of gravity to the behavior of the pendulum becomes evident only as the result of a fairly sophisticated mathematical analysis requiring the genius of a Galileo for its initial formulation. But this "mathematical analysis," be it noted, is full-fledged scientific theory with *bona fide* theorems such as: the longer the suspension of the pendulum, the slower the beat.

In general it may be said that the greater the number of additional principles operative in conjunction with the one under investigation, the more complex the theoretical procedures which are necessary. It is a much more complicated procedure to show theoretically that pendulums should beat more slowly at the equator than at the pole than it is to deduce that pendulums with long suspensions should beat more slowly than those with short ones. This is because in the former situation there must be taken explicitly into consideration the additional principle of the centrifugal force due to the rotation of the earth about its axis.

At the outset of empirical generalization it is often impossible to detect and identify the active scientific principle by mere observation. For example, Newton's principle that all objects attract each other inversely as the squares of the distances separating them was a daring conjecture and one extending much beyond anything directly observable in the behavior of ordinary falling bodies. It is also characteristic that the empirical verification of this epoch-making principle was first secured through the study of careful astronomical measurements rather than through the observation of small falling objects. But the action of gravity in determining the orbits of the planets is even less obvious to ordinary unaided observation than is its rôle in the determination of the behavior of the pendulum. Indeed, this can be detected only by means of the mathematics of the ellipse, i.e., through a decidedly sophisticated theoretical procedure, one which had to await the genius of Newton for its discovery.

Earlier in the chapter it was pointed out that the theoretical outcome, or theorem, derived from a statement of supposed antecedent conditions is assumed in science always to agree with the empirical outcome, provided both procedures have been correctly performed. We must now note the further assumption that if there is disagreement between the two outcomes there must be something wrong with at least one of the principles or rules involved in the

derivation of the theorem; empirical observations are regarded as primary, and wherever a generalization really conflicts with observation the generalization must always give way. When the breakdown of a generalization occurs in this way, an event of frequent occurrence in new fields, the postulates involved are revised if possible so as to conform to the known facts. Following this, deductions as to the outcome of situations involving still other combinations of principles are made; these in their turn are checked against observations; and so on as long as disagreements continue to occur. Thus the determination of scientific principles is in considerable part a matter of symbolic trial-and-error. At each trial of this process, where the antecedent conditions are such as to involve jointly several other presumptive scientific principles, symbolic or theoretical procedure is necessary in order that the investigator may know the kind of outcome to be expected if the supposed principle specially under investigation is really acting as assumed. The empirical procedure is necessary in order to determine whether the antecedent conditions were really followed by the deductively expected outcome. Thus both theoretical and empirical procedures are indispensable to the attainment of the major scientific goal—that of the determination of scientific principles.

HOW THE EMPIRICAL VERIFICATION OF THEOREMS INDIRECTLY SUBSTANTIATES POSTULATES

But how can the empirical verification of the implications of theorems derived from a set of postulates establish the truth of the postulates? In seeking an answer to this question we must note at the outset that absolute truth is not thus established. The conclusion reached in science is not that the postulates employed in the derivation of the empirically verified theorem are thereby shown to be true beyond doubt, but rather that the empirical verification of the theorem has *increased the probability* that the next theorem derived from these postulates in conjunction with a different set of antecedent conditions will also agree with relevant empirical determinations. And this conclusion is arrived at on the basis of chance or probability, i.e., on the basis of a theory of sampling.

The nature of this sampling theory may be best explained by means of a decidedly artificial example. Suppose that by some miracle a scientist should come into possession of a set of postulates none of which had ever been employed, but which were believed

to satisfy the logical criterion of yielding large numbers of empirically testable theorems; that a very large number of such theorems should be deducible by special automatic logical calculation machines; that the theorems, each sealed in a neat capsule, should all be turned over to the scientist at once; and, finally, that these theorems should then be placed in a box, thoroughly mixed, drawn out one at a time, and compared with empirical fact. Assuming that no failures of agreement occurred in a long succession of such comparisons, it would be proper to say that each succeeding agreement would increase the probability that the next drawing from the box would also result in an agreement, exactly as each successive uninterrupted drawing of white marbles from an urn would increase progressively the probability that the next drawing would also yield a white marble. But just as the probability of drawing a white marble will always lack something of certainty even with the best conceivable score, so the validation of scientific principles by this procedure must always lack something of being complete. Theoretical "truth" thus appears in the last analysis to be a matter of greater or less probability. It is consoling to know that this probability frequently becomes very high indeed (3, p. 6).

THE "TRUTH" STATUS OF LOGICAL PRINCIPLES OR RULES

Despite much belief to the contrary, it seems likely that logical (mathematical) principles are essentially the same in their mode of validation as scientific principles; they appear to be merely invented rules of symbolic manipulation which have been found by trial in a great variety of situations to mediate the deduction of existential sequels verified by observation. Thus logic in science is conceived to be primarily a tool or instrument useful for the derivation of dependable expectations regarding the outcome of dynamic situations. Except for occasional chance successes, it requires sound rules of deduction, as well as sound dynamic postulates, to produce sound theorems. By the same token, each observationally confirmed theorem increases the justified confidence in the logical rules which mediated the deduction, as well as in the "empirical" postulates themselves. The rules of logic are more dependable, and consequently less subject to question, presumably because they have survived a much longer and more exacting period of trial than is the case with most scientific postulates. Probably it is because of the widespread and relatively unquestioned ac-

ceptance of the ordinary logical assumptions, and because they come to each individual investigator ready-made and usually without any appended history, that logical principles are so frequently regarded with a kind of religious awe as a subtle distillation of the human spirit; that they are regarded as never having been, and as never to be, subjected to the tests of validity usually applicable to ordinary scientific principles; in short, that they are strictly "self-evident" truths (3, p. 7). As a kind of empirical confirmation of the above view as to the nature of logical principles, it may be noted that both mathematicians and logicians are at the present time busily inventing, modifying, and generally perfecting the principles or rules of their disciplines (6).

SUMMARY

Modern science has two inseparable components—the empirical and the theoretical. The empirical component is concerned primarily with observation; the theoretical component is concerned with the interpretation and explanation of observation. A natural event is explained when it can be derived as a theorem by a process of reasoning from (1) a knowledge of the relevant natural conditions antedating it, and (2) one or more relevant principles called postulates. Clusters or families of theorems are generated, and theorems are often employed in the derivation of other theorems; thus is developed a logical hierarchy resembling that found in ordinary geometry. A hierarchy of interrelated families of theorems, all derived from the same set of consistent postulates, constitutes a scientific system.

Scientific theory resembles argumentation in being logical in nature but differs radically in that the objective of argument is to convince. In scientific theory logic is employed in conjunction with observation as a means of inquiry. Indeed, theoretical procedures are indispensable in the establishment of natural laws. The range of validity of a given supposed law can be determined only by trying it out empirically under a wide range of conditions where it will operate in simultaneous conjunction with the greatest variety and combination of other natural laws. But the only way the scientist can tell from the outcome of such an empirical procedure whether a given hypothetical law has acted in the postulated manner is first to deduce by a logical process what the outcome

of the investigation *should* be if the hypothesis really holds. This deductive process is the essence of scientific theory.

The typical procedure in science is to adopt a postulate tentatively, deduce one or more of its logical implications concerning observable phenomena, and then check the validity of the deductions by observation. If the deduction is in genuine disagreement with observation, the postulate must be either abandoned or so modified that it implies no such conflicting statement. If, however, the deductions and the observations agree, the postulate gains in dependability. By successive agreements under a very wide variety of conditions it may attain a high degree of justified credibility, but never absolute certainty.

REFERENCES

1. HULL, C. L. The conflicting psychologies of learning—a way out. *Psychol. Rev.*, 1935, 42, 491-516.
2. HULL, C. L. Mind, mechanism, and adaptive behavior. *Psychol. Rev.*, 1937, 44, 1-32.
3. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
4. NEWTON, I. *Principia* (trans. by F. Cajori). Berkeley: Univ. Calif. Press, 1934.
5. SPINOZA, B. DE. *Ethic* (trans. by W. H. White and A. H. Stirling). New York: Macmillan Co., 1894.
6. WHITEHEAD, A. N., and RUSSELL, M. A. *Principia mathematica* (Vol. I). London: Cambridge Univ. Press. 1935.

CHAPTER II

Introduction to an Objective Theory of Behavior

Having examined the general nature of scientific theory, we must now proceed to the elaboration of an objective theory as applied specifically to the behavior of organisms (10). Preliminary to this great and complex task it will be well to consider a number of the more general characteristics of organismic behavior, as well as certain difficulties which will be encountered and hazards which ought to be avoided.

THE BASIC FACT OF ENVIRONMENTAL-ORGANISMIC INTERACTION

At the outset of the independent life of an organism there begins a dynamic relationship between the organism and its environment. For the most part, both environment and organism are active; the environment acts on the organism, and the organism acts on the environment (5, p. 2). Naturally the terminal phase of any given environmental-organismic interaction depends upon the activity of each; rarely or never can the activity of either be predicted from knowing the behavior characteristics of one alone. The possibility of predicting the outcome of such interaction depends upon the fact that both environment and organism are part of nature, and as such the activity of each takes place according to known rules, i.e., natural laws.

The environment of an organism may conveniently be divided into two portions—the internal and the external. The external environment may usefully be subdivided into the inanimate environment and the animate or organismic environment.

The laws of the internal environment are, for the most part, those of the physiology of the particular organism. The laws of the inanimate environment are those of the physical world and constitute the critical portions of the physical sciences; they are relatively simple and reasonably well known.

The laws of the organismic environment are those of the behavior of other organisms, especially organisms of the same species as the one under consideration; they make up the primary principles of the behavior, or "social," sciences and are comparatively

complex. Perhaps because of this complexity they are not as yet very well understood. Since in a true or symmetrical social situation only organisms of the same species are involved, the basic laws of the activities of the environment must be the same as those of the organism under consideration. It thus comes about that *the objective of the present work is the elaboration of the basic molar¹ behavioral laws underlying the "social" sciences.*

ORGANISMIC NEED, ACTIVITY, AND SURVIVAL

Since the publication by Charles Darwin of the *Origin of Species* (2) it has been necessary to think of organisms against a background of organic evolution and to consider both organismic structure and function in terms of *survival*. Survival, of course, applies equally to the individual organism and to the species. Physiological studies have shown that survival requires special circumstances in considerable variety; these include optimal conditions of air, water, food, temperature, intactness of bodily tissue, and so forth; for species survival among the higher vertebrates there is required at least the occasional presence and specialized reciprocal behavior of a mate.

On the other hand, when any of the commodities or conditions necessary for individual or species survival are lacking, or when they deviate materially from the optimum, a state of *primary need* is said to exist. In a large proportion of such situations the need will be reduced or eliminated only through the action on the environment of a particular sequence of movements made by the organism. For example, the environment will, as a rule, yield a commodity (such as food) which will mediate the abolition of a state of need (such as hunger) only when the movement sequence corresponds rather exactly to the momentary state of the environment; i.e., when the movement sequence is closely synchronized with the several phases of the environmental reactions. If it is to be successful, the behavior of a hungry cat in pursuit of a mouse must vary from instant to instant, depending upon the movements of the mouse. Similarly if the mouse is to escape the cat, its move-

¹ By this expression is meant the uniformities discoverable among the grossly observable phenomena of behavior as contrasted with the laws of the behavior of the ultimate "molecules" upon which this behavior depends, such as the constituent cells of nerve, muscle, gland, and so forth. The term *molar* thus means coarse or macroscopic as contrasted with molecular, or microscopic.

ments must vary from instant to instant, depending upon the movements of the cat.

Moreover, in a given external environment situation the behavior must often differ radically from one occasion to another, depending on the need which chances to be dominant at the time; e.g., whether it be of food, water, or a mate. In a similar manner the behavior must frequently differ widely from one environmental situation to another, even when the need is exactly the same in each environment; a hungry man lost in a forest must execute a very different sequence of movements to relieve his need from what would be necessary if he were in his home.

It follows from the above considerations that *an organism will hardly survive unless the state of organismic need and the state of the environment in its relation to the organism are somehow jointly and simultaneously brought to bear upon the movement-producing mechanism of the organism.*

THE ORGANIC BASIS OF ADAPTIVE BEHAVIOR

All normal higher organisms possess a great assortment of muscles, usually with bony accessories. These motor organs are ordinarily adequate to mediate the reduction of most needs, provided their contractions occur in the right amount, combination, and sequence. The momentary status of most portions of the environment with respect to the organism is mediated to the organism by an immense number of specialized receptors which respond to a considerable variety of energies such as light waves (vision), sound waves (hearing), gases (smell), chemical solutions (taste), mechanical impacts (touch), and so on. The state of the organism itself (the internal environment) is mediated by another highly specialized series of receptors. It is probable that the various conditions of need also fall into this latter category; i.e., in one way or another needs activate more or less characteristic receptor organs much as do external environmental forces.

Neural impulses set in motion by the action of these receptors pass along separate nerve fibers to the central ganglia of the nervous system, notably the brain. The brain, which acts as a kind of automatic switchboard, together with the remainder of the central nervous system, routes and distributes the impulses to individual muscles and glands in rather precisely graded amounts and sequences. When the neural impulse reaches an effector organ

(muscle or gland) the organ ordinarily becomes active, the amount of activity usually varying with the magnitude of the impulse. The movements thus brought about usually result in the elimination of the need, though often only after numerous unsuccessful trials. But organismic activity is by no means always successful; not infrequently death occurs before an adequate action sequence has been evoked.

It is the primary task of a molar science of behavior to isolate the basic laws or rules according to which various combinations of stimulation, arising from the state of need on the one hand and the state of the environment on the other, bring about the kind of behavior characteristic of different organisms. A closely related task is to understand why the behavior so mediated is so generally adaptive, i.e., successful in the sense of reducing needs and facilitating survival, and why it is unsuccessful on those occasions when survival is not facilitated.

THE NEUROLOGICAL VERSUS THE MOLAR APPROACH

From the foregoing considerations it might appear that the science of behavior must at bottom be a study of physiology. Indeed, it was once almost universally believed that the science of behavior must wait for its useful elaboration upon the development of the subsidiary science of neurophysiology. Partly as a result of this belief, an immense amount of research has been directed to the understanding of the detailed or molecular dynamic laws of this remarkable automatic structure. A great deal has been revealed by these researches and the rate of development is constantly being accelerated by the discovery of new and more effective methods of investigation. Nearly all serious students of behavior like to believe that some day the major neurological laws will be known in a form adequate to constitute the foundation principles of a science of behavior.

In spite of these heartening successes, the gap between the minute anatomical and physiological account of the nervous system as at present known and what would be required for the construction of a reasonably adequate theory of molar behavior is impassable. The problem confronting the behavior theorist is substantially like that which would have been faced by Galileo and Newton had they seriously considered delaying their preliminary formulation of the molar mechanics of the physical world until the

micro-mechanics of the atomic and subatomic world had been satisfactorily elaborated.

Students of the social sciences are presented with the dilemma of waiting until the physico-chemical problems of neurophysiology have been adequately solved before beginning the elaboration of behavior theory, or of proceeding in a provisional manner with certain reasonably stable principles of the coarse, macroscopic or molar action of the nervous system whereby movements are evoked by stimuli, particularly as related to the history of the individual organism.

There can hardly be any doubt that a theory of molar behavior founded upon an adequate knowledge of both molecular and molar principles would in general be more satisfactory than one founded upon molar considerations alone. But here again the history of physical science is suggestive. Owing to the fact that Galileo and Newton carried out their molar investigations, the world has had the use of a theory which was in very close approximation to observations at the molar level for nearly three hundred years before the development of the molecular science of modern relativity and quantum theory. Moreover, it is to be remembered that science proceeds by a series of successive approximations; it may very well be that had Newton's system not been worked out when it was there would have been no Einstein and no Planck, no relativity and no quantum theory at all. It is conceivable that the elaboration of a systematic science of behavior at a molar level may aid in the development of an adequate neurophysiology and thus lead in the end to a truly molecular theory of behavior firmly based on physiology.

It happens that a goodly number of quasi-neurological principles have now been determined by careful experiments designed to trace out the relationship of the molar behavior of organisms, usually as integrated wholes, to well-controlled stimulus situations. Many of the more promising of these principles were roughly isolated in the first instance by the Russian physiologist, Pavlov, and his pupils, by means of conditioned-reflex experiments on dogs. More recently extensive experiments in many laboratories in this country with all kinds of reactions on a wide variety of organisms, including man, have greatly extended and rectified these principles and shown how they operate jointly in the production of the more complex forms of behavior. Because of the pressing nature of behavior problems, both practical and theoretical aspects of be-

havior science are, upon the whole, being developed according to the second of the two alternatives outlined above. For these reasons the molar approach is employed in the present work.

In this connection it is to be noted carefully that *the alternatives of microscopic versus macroscopic, and molecular versus molar, are relative rather than absolute*. In short, there are degrees of the molar, depending on the coarseness of the ultimate causal segments or units dealt with. Other things equal, it would seem wisest to keep the causal segments small, to approach the molecular, the fine and exact substructural details, just as closely as the knowledge of that substructure renders possible. There is much reason to believe that the seeming disagreements among current students of behavior may be largely due to the difference in the degree of the molar at which the several investigators are working. Such differences, however, do not represent fundamental disagreements. In the end the work of all who differ only in this sense may find a place in a single systematic structure, the postulates or primary assumptions of those working at a more molar level ultimately appearing as theorems of those working at a more molecular level.

THE RÔLE OF INTERVENING VARIABLES IN BEHAVIOR THEORY

Wherever an attempt is made to penetrate the invisible world of the molecular, scientists frequently and usefully employ logical constructs, intervening variables, or symbols to facilitate their thinking. These symbols or *X*'s represent entities or processes which, if existent, would account for certain events in the observable molar world. Examples of such postulated entities in the field of the physical sciences are electrons, protons, positrons, etc. A closely parallel concept in the field of behavior familiar to everyone is that of *habit* as distinguished from habitual action. The habit presumably exists as an invisible condition of the nervous system quite as much when it is not mediating action as when habitual action is occurring; the habits upon which swimming is based are just as truly existent when a person is on the dance floor as when he is in the water.

In some cases there may be employed in scientific theory a whole series of hypothetical unobserved entities; such a series is presented by the hierarchy of postulated physical entities: molecule, atom, and electron, the molecule supposedly being constituted of atoms and the atom in its turn being constituted of electrons.

A rough parallel to this chain of hypothetical entities from the physical sciences will be encountered in the present system of behavior theory. For the above reasons the subject of symbolic constructs, intervening variables, or hypothetical entities which are not directly observable requires comment (6, p. 3 ff.).

Despite the great value of logical constructs or intervening variables in scientific theory, their use is attended with certain difficulties and even hazards. At bottom this is because the presence and amount of such hypothetical factors must always be determined indirectly. But once (1) the dynamic relationship existing between the amount of the hypothetical entity (X) and some antecedent determining condition (A) which can be directly observed, and (2) the dynamic relationship of the hypothetical entity to some third consequent phenomenon or event (B) which also can be directly observed, become fairly well known, the scientific hazard largely disappears. The situation in question is represented in Figure 1. When a hypothetical dynamic entity, or even

$$A \longrightarrow f \longrightarrow (X) \longrightarrow f \longrightarrow B$$

FIG. 1. Diagrammatic representation of a relatively simple case of an intervening variable (X) not directly observable but functionally related (f) to the antecedent event (A) and to the consequent event (B), both A and B being directly observable. When an intervening variable is thus securely anchored to observables on both sides it can be safely employed in scientific theory.

a chain of such entities each functionally related to the one logically preceding and following it, is thus securely anchored on both sides to observable and measurable conditions or events (A and B), the main theoretical danger vanishes. This at bottom is because under the assumed circumstances no ambiguity can exist as to when, and how much of, B should follow A .

THE OBJECTIVE VERSUS THE SUBJECTIVE APPROACH TO BEHAVIOR THEORY

If the circumstances sketched above as surrounding and safeguarding the use of hypothetical entities are not observed, the grossest fallacies may be committed. The painfully slow path whereby man has, as of yesterday, begun to emerge into the truly scientific era is littered with such blunders, often tragic in their

practical consequences. A pestilence or a hurricane descends upon a village and decimates the population. The usual hypothesis put forward by primitive man (and many others who think themselves not at all primitive) to explain the tragic event (*B*) is that some hypothetical spirit (*X*) has been angered by the violation (*A*) of some tribal taboo on the part of one or more inhabitants of the village. Unfortunately this mode of thinking is deeply ingrained in most cultures, not excepting our own, and it even crops up under various disguises in what purports to be serious scientific work.

Perhaps as good an example of such a fallacious use of the intervening variable as is offered by recent scientific history is that of the *entelechy* put forward by Hans Driesch as the central concept in his theory of vitalism (*§*). Driesch says, for example:

A *supreme* mind, conversant with the inorganic facts of nature and knowing all the intensive manifoldness of all entelechies and psychoids . . . would be able to predict the individual history of the latter, would be able to predict the actions of any psychoid with absolute certainty. *Human* mind, on the other hand, is not able to predict in this way, as it does not know entelechy before its manifestation, and as the material conditions of life, which alone the mind of man *can* know . . . in its completeness, are not the only conditions responsible for organic phenomena. (*§*, p. 249.)

Driesch's entelechy (*X*) fails as a logical construct or intervening variable not because it is not directly observable (though of course it is not), but because the general functional relationship to antecedent condition *A* and that to consequent condition *B* are *both* left unspecified. This, of course, is but another way of saying that the entelechy and all similar constructs are essentially metaphysical in nature. As such they have no place in science. *Science has no use for unverifiable hypotheses.*

A logically minded person, unacquainted with the unscientific foibles of those who affect the scientific virtues, may naturally wonder how such a formulation could ever mediate a semblance of theoretical prediction and thus attain any credence as a genuinely scientific theory. The answer seems to lie in the inveterate animistic or anthropomorphic tendencies of human nature. The entelechy is in substance a spirit or daemon, a kind of vicarious ghost. The person employing the entelechy in effect says to himself, "If I were the entelechy in such and such a biological emergency, what would I do?" Knowing the situation and what is required to meet the emergency, he simply states what he knows to be required as a solution, and he at once has in this statement what purports to be

a scientific deduction! He has inadvertently substituted himself in place of the construct and naïvely substituted his knowledge of the situation for the objective rules stating the functional relationships which *ought* to subsist between *A* and *X* on the one hand, and between *X* and *B* on the other.

This surreptitious substitution and acceptance of one's knowledge of what needs to be done in a biological emergency for a theoretical deduction is the essence of what we shall call *anthropomorphism*, or the *subjective*, in behavior theory. After many centuries the physical sciences have largely banished the subjective from their fields, but for various reasons this is far less easy of accomplishment and is far less well advanced in the field of behavior. The only known cure for this unfortunate tendency to which all men are more or less subject is a grim and inflexible insistence that all deductions take place according to the explicitly formulated rules stating the functional relationships of *A* to *X* and of *X* to *B*. This latter is the essence of the scientifically *objective*. A genuinely scientific theory no more needs the anthropomorphic intuitions of the theorist to eke out the deduction of its implications than an automatic calculating machine needs the intuitions of the operator in the determination of a quotient, once the keys representing the dividend and the divisor have been depressed.

Objective scientific theory is necessary because only under objective conditions can a principle be tested for soundness by means of observation. The basic difficulty with anthropomorphic subjectivism is that what appear to be deductions derived from such formulations do not originate in rules stating postulated functional relationships, but rather in the intuitions of the confused thinker. Observational check of such pseudo-deductions may verify or refute these intuitions, but has no bearing on the soundness of any scientific principles whatever; such verifications or refutations might properly increase the reputation for accurate prophecy of the one making such intuitive judgments, but a prophet is not a principle, much less a scientific theory.

OBJECTIVISM VERSUS TELEOLOGY

Even a superficial study of higher organisms shows that their behavior occurs in cycles. The rise of either a primary or a secondary need normally marks the beginning of a behavior cycle, and the abolition or substantial reduction of that need marks its

end. Some phase of the joint state of affairs resulting from the environmental-organismic interaction at the end of a behavior cycle is customarily spoken of as a goal. Our usual thoughtless custom is to speak of cycles of behavior by merely naming their outcome, effect, or end result, and practically to ignore the various movements which brought this terminal state about. Guthrie has expressed this tendency more aptly than anyone else (4, p. 1). We say quite naturally that a man catches a fish, a woman bakes a cake, an artist paints a picture, a general wins a battle. The end result of each angling exploit, for example, may be in some sense the same but the actual movements involved are perhaps never exactly the same on any two occasions; indeed, neither the angler nor perhaps anyone else knows or could know in their ultimate detail exactly what movements were made. It is thus inevitable that for purposes of communication we designate behavior sequences by their goals.

Now for certain rough practical purposes the custom of naming action sequences by their goals is completely justified by its convenience. It may even be that for very gross molar behavior it can usefully be employed in theory construction, provided the theorist is alert to the naturally attendant hazards. These appear the moment the theorist ventures to draw upon his intuition for statements concerning the behavior (movements) executed by the organism between the onset of a need and its termination through organismic action. Pseudo-deductions on the basis of intuition born of intimate knowledge are so easy and so natural that the tendency to make them is almost irresistible to most persons. The practice does no harm if the theorist does not mistake this subjective intuitional performance for a logical deduction from an objective theory, and attribute the success of his intuitions to the validity of the theoretical principles.

An ideally adequate theory even of so-called purposive behavior ought, therefore, to begin with colorless movement and mere receptor impulses as such, and from these build up step by step both adaptive behavior and maladaptive behavior. The present approach does not deny the molar reality of purposive acts (as opposed to movement), of intelligence, of insight, of goals, of intents, of strivings, or of value; on the contrary, we insist upon the genuineness of these forms of behavior. We hope ultimately to show the logical right to the use of such concepts by deducing them as secondary principles from more elementary objective primary

principles. Once they have been derived we shall not only understand them better but be able to use them with more detailed effectiveness, particularly in the deduction of the movements which mediate (or fail to mediate) goal attainment, than would be the case if we had accepted teleological sequences at the outset as gross, unanalyzed (and unanalyzable) wholes.

"EMERGENTISM" A DOCTRINE OF DESPAIR

Perhaps the very natural and economical mode of communication whereby we speak of the terminal or goal phases of action, largely regardless of the antecedent movements involved, predisposes us to a belief in *teleology*. In its extreme form teleology is the name of the belief that the *terminal* stage of certain environmental-organismic interaction cycles somehow is at the same time one of the *antecedent* determining conditions which bring the behavior cycle about. This approach, in the case of a purposive behavior situation not hitherto known to the theorist, involves a kind of logical circularity: to deduce the outcome of any behavioral situation in the sense of the deductive predictions here under consideration, it is necessary to know all the relevant antecedent conditions, but these cannot be determined until the behavioral outcome has been deduced. In effect this means that the task of deduction cannot begin until after it is completed! Naturally this leaves the theorist completely helpless. It is not surprising that the doctrine of teleology leads to theoretical despair and to such pseudo-remedies as vitalism and *emergentism*.

Emergentism, as applied to organismic behavior, is the name for the view that in the process of evolution there has "emerged" a form of behavior which is ultimately unanalyzable into logically more primitive elements—behavior which cannot possibly be deduced from any logically prior principles whatever. In particular it is held that what is called goal or purposive behavior is of such a nature, that it cannot be derived from any conceivable set of postulates involving mere stimuli and mere movement (8, pp. 7-8; 7, pp. 26-27).

On the other hand, many feel that this defeatist attitude is not only unwholesome in that it discourages scientific endeavor, but that it is quite unjustified by the facts. The present writer shares this view. Therefore a serious attempt will ultimately be made to show that these supposedly impossible derivations are actually pos-

sible; in some cases they will be shown to be quite easy of accomplishment.

A SUGGESTED PROPHYLAXIS AGAINST ANTHROPOMORPHIC SUBJECTIVISM

As already suggested, one of the greatest obstacles to the attainment of a genuine theory of behavior is anthropomorphic subjectivism. At bottom this is because we ourselves are so intimately involved in the problem; we are so close to it that it is difficult to attain adequate perspective. For the reader who has not hitherto struggled with the complex but fascinating problems of behavior theory, it will be hard to realize the difficulty of maintaining a consistently objective point of view. Even when fully aware of the nature of anthropomorphic subjectivism and its dangers, the most careful and experienced thinker is likely to find himself a victim to its seductions. Indeed, despite the most conscientious effort to avoid this it is altogether probable that there may be found in various parts of the present work hidden elements of the anthropomorphically subjective.

One aid to the attainment of behavioral objectivity is to think in terms of the behavior of subhuman organisms, such as chimpanzees, monkeys, dogs, cats, and albino rats. Unfortunately this form of prophylaxis against subjectivism all too often breaks down when the theorist begins thinking what he would do if he were a rat, a cat, or a chimpanzee; when that happens, all his knowledge of his own behavior, born of years of self-observation, at once begins to function in place of the objectively stated general rules or principles which are the proper substance of science.

A device much employed by the author has proved itself to be a far more effective prophylaxis. This is to regard, from time to time, the behaving organism as a completely self-maintaining robot, constructed of materials as unlike ourselves as may be. In doing this it is not necessary to attempt the solution of the detailed engineering problems connected with the design of such a creature. It is a wholesome and revealing exercise, however, to consider the various general problems in behavior dynamics which must be solved in the design of a truly self-maintaining robot. We, in common with other mammals, perform innumerable behavior adaptations with such ease that it is apt never to occur to us that any problem of explanation exists concerning them. In many such

seemingly simple activities lie dynamical problems of very great complexity and difficulty.

A second and closely related subjective tendency against which the robot concept is likely to prove effectively prophylactic is that to the *reification* of a behavior function. To reify a function is to give it a name and presently to consider that the name represents a thing, and finally to believe that the thing so named somehow *explains* the performance of the function. We have already seen an example of this unfortunate tendency in Driesch's entelechy. The temptation to introduce an entelechy, soul, spirit, or daemon into a robot is slight; it is relatively easy to realize that *the introduction of an entelechy would not really solve the problem of design of a robot because there would still remain the problem of designing the entelechy itself, which is the core of the original problem all over again.* The robot approach thus aids us in avoiding the very natural but childish tendency to choose easy though false solutions to our problems, by removing all excuses for not facing them squarely and without evasion.

Unfortunately it is possible at present to promise an explanation of only a portion of the problems encountered in the infinitely complex subject of organismic behavior. Indeed, it is no great exaggeration to say that the present state of behavior theory resembles one of those pieces of sculpture which present in the main a rough, unworked block of stone with only a hand emerging in low relief here, a foot or thigh barely discernible there, and elsewhere a part of a face. The undeveloped state of the behavior sciences suggested by this analogy is a source of regret to the behavior theorist but not one of chagrin, because incompleteness is characteristic even of the most advanced of all theoretical sciences. From this point of view the difference between the physical and the behavioral sciences is one not of kind but of degree—of the relative amount of the figure still embedded in the unhewn rock. There is reason to believe that the relative backwardness of the behavior sciences is due not so much to their inherent complexity as to the difficulty of maintaining a consistent and rigorous objectivism.

SUMMARY

The field of behavior theory centers primarily in the detailed interaction of organism and environment. The basic principles of organismic behavior are to be viewed against a background of

organic evolution, the success or failure of the evolutionary process being gauged in terms of survival. Individual and species survival depend upon numerous optimal physiological conditions; when one of these critical conditions deviates much from the optimum, a state of primary need arises. Need reduction usually comes about through a particular movement sequence on the part of the organism. Such sequences depend for their success jointly upon the nature of the need and the nature and state of the environment.

The condition of organismic need and the status of the environment evoke from specialized receptors neural impulses which are brought to bear jointly on the motor organs by the central ganglia of the nervous system acting as an automatic switchboard. The primary problem of behavior theory is to discover the laws according to which this extraordinarily complex process occurs. Students of behavior have resorted to the coarse, or "molar," laws of neural activity as revealed by conditioned-reflex and related experiments, rather than to the "molecular" results of neurophysiology, because the latter are not yet adequate.

Perhaps partly as the result of this molar approach, it is found necessary to introduce into behavior theory numerous logical constructs analogous to molecules and atoms long used in the physical sciences. All logical constructs present grave theoretical hazards when they are not securely anchored to directly observable events both as antecedents and consequences by definite functional relationships. Under conditions of unstated functional relationships the naïve theorist is tempted to make predictions on the basis of intuition, which is anthropomorphic subjectivism. The derivation of theoretical expectations from explicitly stated functional relationships is the objective method. Experimental agreement with expectations can properly validate theoretical principles only when objective procedures are employed.

Some writers believe that there is an impassable theoretical gulf between mere muscle contraction and the attainment of goals; that the latter are "emergents." This doctrine of despair grows naturally out of the doctrine of teleology. The present treatise accepts neither teleology nor its pessimistic corollary. Goals, intents, intelligence, insight, and value are regarded not only as genuine but as of the first importance. Ultimately an attempt will be made to derive all of these things objectively as secondary phenomena from more elementary objective conditions, concepts, and principles.

NOTES

Operational Definitions and Intervening Variables

In 1938 Bridgman, a physicist whose chief research activities have been concerned with the empirical determination of various physical phenomena under very great pressures, wrote a book (1) in which he made an acute examination of the use of various concepts in current physical theory, particularly those representing intervening variables. The cure which he recommended for such abuses as he found was the scrupulous recognition of the operations carried out by the experimentalists as a means to the making of the observations and measurements of the observable events (*A* and *B*, Figure 1). This, as we saw above, has special significance for the science of behavior, which is so prone to the subjective use of intervening variables. Quite naturally and properly, Bridgman's work has greatly impressed many psychologists. Unfortunately his emphasis upon the operations which are the means whereby the observations and measurements in question become possible has led many psychologists to mistake the means for the end. The point here to be emphasized is that while observations must be considered in the context of the operations which make them possible, the central factor in the situation is *what is observed*. The moral of Bridgman's treatise is that the intervening variable (*X*) is never directly observed but is an inference based on the observation of something else, and that the inference is critically dependent upon the experimental manipulations (operations) which lead to the observations. An emphasis on operations which ignores the central importance of the dependent observations completely misses the virtue of what is coming to be known as operationism.

The Subjective Versus the Objective in Behavior Theory

The critical characteristic of the subjective as contrasted with the objective is that the subjective tends to be a private event, whereas the objective is a public event, i.e., an event presumed to be independently observable by many persons. Thus the perceptual experience or conscious feeling of a person when stimulated by light rays of a certain wave length is said to be a private or subjective event, whereas the light rays themselves, or the overt behavior of another person in response to the impact of the light rays, is said to be a public or objective event.

A typical case of subjectivism in the field of theory, on the other hand, is one in which the alleged theorist asserts, and even believes, that he has deduced a proposition in a logical manner, whereas in fact he has arrived at it by mere anthropomorphic intuition. The subjectivism of behavior theory is thus dependent upon a kind of privacy, but one quite different from that of perceptual consciousness or experience. The subjective aspect of experience is dependent upon the private nature of the process hidden within the body of the *subject*; subjectivism in the field of behavior theory, on the other hand, is dependent upon the private nature of the processes within the body of the *theorist*, whereby he attempts to explain the behavior of the subject. A theory becomes objective when the primary assumptions and the logical steps whereby these assumptions lead to further propositions (theorems) are exhibited to public observation and so make possible a kind of repetition of the logical process by any other person.

Propositions originating in private intuitions masquerading as unstated logical processes are, of course, not theoretical material at all, and have no proper place in science.

Historical Note Concerning the Concept of Molar Behavior and of the Intervening Variable

The important concept of molar, as contrasted with molecular, behavior was introduced into psychology in 1931 by E. C. Tolman. The present writer has taken over the concept substantially as it appears in Tolman's well-known book (8).

The explicit introduction into psychology of the equally important concept of the intervening variable is also due to Professor Tolman; its first and best elaboration was given in his address as President of the American Psychological Association, delivered at Minneapolis, September 3, 1937 (9).

REFERENCES

1. BRIDGMAN, P. W. *The logic of modern physics*. New York: Macmillan, 1938.
2. DARWIN, C. *Origin of species*. New York: Modern Library, 1936.
3. DRIESCH, H. *The science and philosophy of the organism*, second ed. London: A. and C. Black, 1929.
4. GUTHRIE, E. R. Association and the law of effect. *Psychol. Rev.*, 1940, 47, 127-148.
5. HULL, C. L. Conditioning: outline of a systematic theory of learning (Chapter II in *The psychology of learning*, Forty-First Yearbook of the National Society for the Study of Education, Part II). Bloomington, Ill.: Public School Publishing Co., 1942.
6. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
7. KOFFKA, K. *Principles of Gestalt psychology*. New York: Harcourt, Brace and Co., 1935.
8. TOLMAN, E. C. *Purposive behavior in animals and men*. New York: Century Co., 1932.
9. TOLMAN, E. C. The determiners of behavior at a choice point. *Psychol. Rev.*, 1938, 45, 1-41.
10. WATSON, J. B. *Psychology from the standpoint of a behaviorist*, second ed. Philadelphia: J. B. Lippincott Co., 1924.

CHAPTER III

Stimulus Reception and Organism Survival

The intimate relation of the motility of organisms to survival has repeatedly been emphasized (p. 17). It has also been pointed out (p. 18) that if the action of organisms is to facilitate survival, movement must vary in an intimate manner not only with the state of the need but with the exact state of both the internal and the external environment at the instant of action occurrence. This means that the survival of the organism usually requires a precise integration of the animal's motor organs with its environment. The means whereby the various stimulating energies of the environment are mediated to the nervous system, the central integrating mechanism of organisms, must therefore be examined.

SOME TERMINOLOGICAL CLARIFICATIONS

Owing in part to the historical contamination of behavior science by metaphysical speculation, certain ambiguities and misunderstandings have arisen concerning the meanings of terms commonly employed to indicate phenomena associated with receptor activity and functioning. We shall accordingly state our own use of these terms, employing the visual receptor for purposes of illustration.

Light is believed by most physicists to be a wave phenomenon, the different wave lengths (or frequencies) being reflected from the surfaces of objects. Consider, for example, the large, red celluloid die represented in Figure 2, against a background of black velvet. The die itself we shall call a *stimulus object*. From the surface of this stimulus object, wave lengths of approximately 650 millimicrons, say, are reflected in all directions except where opaque obstructions exist. From the white spots, waves of all lengths are reflected. The amount of the light reflected in a given direction will vary jointly with the angle of the surface involved and the direction of the light source. Any and all of the rays of light reflected from the die are *potential stimuli*, depending upon whether or not a responsive receptor chances to be in such a position as to

receive them; in the latter case the light ray becomes an *actual stimulus* (see Figure 2).

Suppose, now, that the die were to be rotated slowly on its vertical axis as it stands in Figure 2. It is clear that the retina

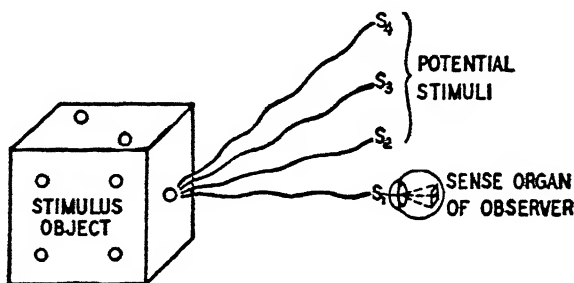


FIG. 2. Diagrammatic representation of a stimulus object, a sheaf of potential stimuli, an actual stimulus (S_1), and the sense organ of the observer upon which the actual stimulus impinges (S).

would receive the impact of a gradually changing pattern or combination of light waves, each of the infinite number of angles resulting from the rotation reflecting a different configuration of stimulated points on the retina. Such a series of compound stimulations is called a *stimulus continuum*.

SOME TYPICAL RECEPTORS AND THEIR ADAPTIVE FUNCTIONS

The processes of organic evolution have solved the primary problem of mediating to the organism the differential nature and state of the environment by developing specially differentiated organs each of which is normally and primarily stimulated only by a limited range of environmental energy. The normal individuals of the higher mammalian species possess receptors which respond to the stimulation of most forms of energy critically active during the period of evolution. Generally speaking, a distinct receptor organ is available for each energy type. In this way the several vitally important energy manifestations of the environment are *qualitatively* differentiated. We proceed now to a brief survey of the more important of these receptors, with special reference to the biological function performed by each.

Biologically, one of the most primitive and necessary of the receptors is that responsive to contact or mechanical pressure.

The end organs are appropriately located in the skin or membranes susceptible to contact. An interesting extension of the contact receptor system beyond the surface of the organism is brought about by the fact that on the hairy parts the touch receptor is placed at the base of the hairs in such a way that when the shaft of hair is moved the receptor is activated. Since an object is likely to touch a hair before it reaches the skin, we have here a beginning of distance reception. This adaptive device is developed still further in certain organisms such as cats and rats in the long and relatively stiff hairs which extend outward from the face, mostly in the region of the nose. These extended or indirect touch receptors become very useful distance receptors in darkness, even though their range be small.

One of the many necessities of the mammalian body is the maintenance of an optimum temperature. This means that receptors must be provided which will yield differential neural impulses when the environment is such as to cool the skin below, or to raise it above, a certain temperature. Instead of giving us a single thermometer to act on the thermostat principle, nature seems to have evolved *two* receptors, one for temperatures above about 33.5 C. degrees, and another for temperatures below about 32.5 C. degrees, depending somewhat upon circumstances. The receptor organs for temperature have not yet been determined with complete certainty (6, p. 1053 ff.).

Perhaps the most imperative receptor need of the organism is to have organs responsive to a state of injury within the internal environment, i.e., to the destruction of tissue or to situations which if intensified or long continued would result in the destruction of tissue. The process of organic evolution has provided the higher organisms with an abundant supply of such end organs. They are sometimes called nociceptors. The hollow organs of the viscera are provided with nociceptors which are activated by persistent distention. The organs responsive to tissue injury, if any, also have not yet been determined with certainty (6, p. 1065 ff.).

In order that the organism may behave differentially to good and bad food, it is clear that receptors yielding differential neural reactions to such substances must be provided. Nature has solved this problem by evolving chemical receptors for liquids—the gustatory receptor organs, or taste buds, located mostly along the sides and at the back of the tongue. The location of these end organs in the mouth is highly adaptive. They will not be activated by

sapid substances unless the latter are about to be ingested, yet the process of ingestion will not have gone so far at the time of end organ activation that rejection will not be easily possible (6, p. 1005 ff.).

The smell or olfactory receptors are activated by certain gases. Quite appropriately the olfactory end organs are found in an upper chamber of the nose where they will be in contact with eddies from a constantly changing sample of the aerial environment of the organism, incidental to the breathing activities (6, p. 992). The olfactory receptor is in a favorable position for guarding the alimentary canal against undesirable substances and for guiding to suitable food substances. Since gases characteristic of a substance are apt to surround it for some distance, the receptor's response to these may evoke rejection before the substance even enters the mouth (6, p. 992 ff.).

THE RECEPTION OF MOVEMENT

An exceedingly important sense mediating one aspect of the internal environment is that known as proprioception, or kinaesthesia. The receptors of this sense organ lie mainly in the muscles and joint capsules. Since they are stimulated by movements of either the muscles or the joints, these receptors become of special value in mediating all forms of activity involving a considerable degree of muscular coördination (6, p. 1072 ff.). Ultimately we shall see reason to believe that this seemingly humble and obscure receptor plays an indispensable rôle in the most complex and subtle activities ever evolved by nature—those of symbolic behavior or thought.

In the continued survey of movement reception we find that nature has evolved a remarkable organ which gives differential and characteristic receptor response to angular or progressive motions of the head, whether the motion be active or passive. The ultimate receptors lie within the labyrinth (6, p. 204 ff.). This structure consists in the main of three small semi-circular canals placed at right angles to each other within the head and intimately related topologically to the internal ear. When the head is turned through an angle or moved progressively, the receptor organs within the labyrinth are stimulated in a distinctive manner, in this way initiating characteristic neural impulses. The particular receptor organs influenced by a given turning movement of the head will

depend largely upon the position in which the head is supported by the neck muscles. Thus adaptive reactions to angular or progressive movements of the body as a whole must be based on the combination or pattern (see p. 44 ff. and p. 349 ff.) of the neural impulses arising in the labyrinth and those arising in the proprioceptive end organs in the neck and other parts of the body. It is accordingly not accidental that the nerve fibers extending to the brain from both the labyrinth and the muscles largely enter the same general portion of the brain, namely, the cerebellum.

THE RECEPTION OF THE SPATIAL RELATIONSHIPS

Even a cursory observation of organisms shows that their survival depends upon their behavior being coördinated not only to the objects and substances in their environment, but to the distances and directions of these from the organism and from each other. We have already noted an exceedingly crude distance receptor in hairs, some of which are specialized for this function. Moreover, the proprioceptive and labyrinthine receptors are clearly connected with active and passive movement, and so are closely related to distance and direction.

The olfactory receptor must also be mentioned in this connection. Certain bodies, substances, and organisms give off gases, and these gases spontaneously diffuse through the atmosphere. In general the concentration of these gases will be greater, and so the intensity of receptor response will be the more intense, the closer the gas-emitting object is to the receptor. Intensity of olfactory receptor response accordingly becomes a basis for the mediation to the organism of a spatial relationship. The fact that gases are disseminated by means of air currents as well as by conduction has both advantages and disadvantages from the point of view of organism adaptation. If the air current reaches the receptor after passing near the redolent object, activation will occur, but if not, the gas will never reach the receptor and so the olfactory end organ will fail as a distance receptor.

A much more dependable distance receptor has been developed incidental to the evolution of a receptor responsive to vibrations in the air. The ultimate end organs of this receptor lie in the cochlea of the inner ear. The reason the sense of hearing is a good distance receptor is that any vibrating object sets the neighboring air into vibration at the same rate, and this vibration is

propagated to adjacent air with gradually diminishing amplitude at the rate of a little over a thousand feet per second. This means that if an auditory receptor sensitive to a frequency of 100 vibrations per second begins sending neural impulses into the nervous system, an object vibrating at the rate of 100 per second must be somewhere in the external environment.

The differential response of the auditory receptors to the particular distance and direction of the vibrating object is mediated in a striking manner. Other things equal, the intensity of the vibratory impact on the end organ varies inversely with the distance. Unfortunately, this is a somewhat ambiguous criterion of distance, since the intensity of vibratory impact is also dependent upon the vibrational amplitude of the originating object.

The matter of the receptor response to direction is more complex. This seems to be jointly dependent upon two factors. Since there is an ear at each side of the head, the ear which is turned toward the origin of an auditory vibration receives a somewhat more intense vibratory impact than does the other ear. A second factor, also dependent upon the double nature of the auditory receptor, is that the phases of the air waves vary a little at the respective ears when one ear is turned toward the source as compared with the identity of the phase occurring when both ears are equally near the source. Moreover, the extent and nature of this dissimilarity in wave phase vary continuously as the head is rotated through 180 degrees from a position in which one ear is turned directly toward the vibration source.

The queen of the senses and the distance and direction receptor *par excellence* is the eye. The ultimate end organs of vision are microscopic rods and cones imbedded in the retina. The accessory mechanisms of the eye such as the iris, the lens, and the various humors are merely means for bringing the light energy reflected from the environment into adequate contact with the retina.

Owing to the action of the lens system of the eye, limited portions of the external environment are projected with considerable fidelity upon the retina. In this way the spatial pattern of light frequencies reflected from environmental objects is presented directly to the retina, giving precise and characteristic neural responsiveness corresponding to each element of the pattern. This incomparable sense organ, with its responsiveness to the different intensities of the various wave lengths of light, thus furnishes the

receptor basis for an almost unlimited degree of differential adaptive reaction to distinct stimulus objects and situations.

Passing now to the matter of visual distance reception proper, we find that the physics of light furnishes a reliable foundation in the fact that the image of any given object as projected upon the retina varies inversely in size with the distance of the object from the eye. There is a slight ambiguity here in that the size of the image is also dependent upon the size of the object itself, which may vary considerably.

On the retina directly back of the pupil is a point of especially clear vision called the *fovea*. Other things equal, detailed sensitivity to visual patterns grows progressively less with distance from the fovea toward the periphery of the retina. Now it happens that the eye, unlike the ear, is a very mobile organ. It thus comes about that organisms readily learn to roll the eye in its socket in such a way that the image of an object of importance for adaptive reaction will fall on the fovea of each eye. This is called *fixation*. The movements of each eye in its socket are produced by the action of a set of six small external muscles. It is clear that the tension of the muscles which turn the eye in its socket, in conjunction with the tension of the muscles of the neck, must yield a combination or pattern of proprioceptive neural impulses unambiguously correlated with the *direction* of the object fixated.

Just as the doubleness of the hearing organ facilitates the reception of auditory distance cues, so the fact that we have two eyes aids in the reception of visual distance cues. Since the eyes are some inches apart, fixation on a single point near at hand produces a certain amount of *convergence*, and the closer the object, the greater the convergence. This may easily be verified by asking a friend to look at your finger as you move it forward and backward from six to eighteen inches before his face. As the eyes turn inward, the tension of the muscles performing this action varies inversely with the distance of the object fixated. The proprioceptive receptors in certain of these muscles are accordingly stimulated to an extent which varies inversely with the distance of the object. In this roundabout way, involving the joint action of vision and proprioception, the differential sensory reception of the distance of objects within from 60 to 100 feet is accomplished with considerable precision.

THE MEDIATION OF TEMPORAL RELATIONSHIPS

Careful observation of the conditions to which organisms must adapt themselves if they are to survive shows that in addition to the qualitative and spatial characteristics of the environment, the timing of behavior is frequently very important. The processes of organic evolution appear to have solved this problem in an even more roundabout and obscure manner than some of the receptor problems hitherto considered. In fact, the temporal characteristics of environmental events seem to be mediated without the action of any special receptor organ at all.

It will be shown later (Figures 3 and 4) that the frequency of neural impulses emitted by a stimulated receptor undergoes a characteristic change during the continued action of the unchanged stimulating energy. There is also reason to believe that the effects of a stimulation which has ceased, persist for some time (p. 41), meanwhile undergoing progressive and consistent diminution. These changes in the neural responses to stimulation, almost purely as a function of time, are believed to furnish organisms with an adequate basis for timing their movements both during the continuance of a critical stimulus and after its termination.

THE PRIMARY PRINCIPLE OF STIMULATION

The first step in the neural mediation of the state of both the internal and the external environment to the effectors of the organism is dependent upon the principle of stimulation or excitation. The critical characteristic of this principle is that a small amount of energy acting on some specialized structure will release into activity potential energy from some other source, often in relatively large amounts. A familiar example is the trigger action of a gun. The projectile is impelled by the energy stored in the explosive charge; the pressure of the finger on the trigger merely serves to initiate the explosive action which, once started, is self-propagated.

The principle of stimulation is operative at several points in the integrative apparatus of the mammalian organism, two of which we need to consider here. The first is the action of an energy source, such as light, upon a receptor organ such as the eye, which initiates a self-propagating impulse in an afferent nerve fiber. The second is the action of an efferent neural impulse when

it impinges upon a muscle fiber. In this second case there results a release of energy stored in the cell which takes the form of longitudinal contraction (2).

QUALITATIVE VERSUS QUANTITATIVE RECEPTOR ANALYSIS OF ENVIRONMENTAL ENERGIES

Modern neurophysiological studies have shown that the neural discharges initiated by the stimulation of all receptors are substantially alike—a series of discrete waves (Figure 3). This seems to

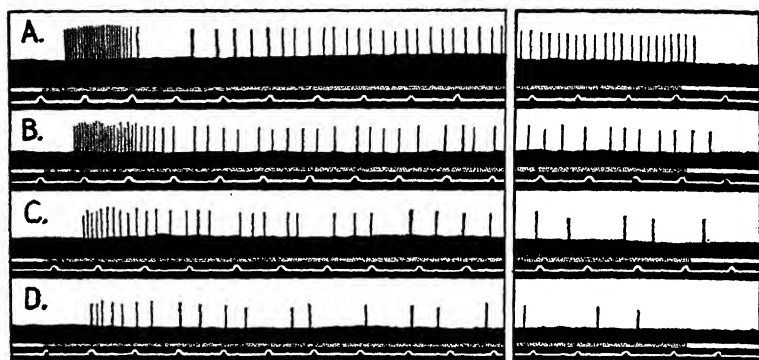


FIG. 3. Reproduction of a photographic record of the action potentials from a single optic-nerve fiber of a horseshoe crab when the end organ was stimulated with different intensities of light. The gaps in the records represent the passage of 2.8, 1.4, 4.5, and 3.3 seconds respectively. In record A, stimulation was .1 light unit; in B, .01; in C, .001; and in D, .0001. The duration of the action of the light on the end organ is indicated by the shadowy line just above the time line in each record. Note that the stronger the stimulation, the more rapid the neural impulses; and the longer the duration of the light, the slower the impulse emission. Note, also, that the light acts for an appreciable time before any neural impulses are emitted (latency); that this latency is shorter, the more intense the light energy; and that usually there are a few neural impulses emitted after the termination of the light. This is known as the after-discharge. (After Graham, 6, p. 830.)

indicate that the only means whereby qualitative differentiation of environmental energies can take place in the nervous system must lie in the differentiation of the nerve fibers which transmit the impulses. Such an arrangement clearly provides a basis whereby the automatic switchboard activities of the central nervous system may route the impulses initiated by qualitatively distinct stimuli (distinct forms of energy) to different muscles and muscle combinations.

But if behavior is to be thoroughly adaptive, it must vary with the quantitative differences in environmental energies as well as with their qualitative differences. Observation indicates that higher organisms do, in fact, react differentially to varying intensities of exactly the same forms of activating energy. If the radio gives forth too weak a tone we turn the volume knob in one direction, and if it gives forth too strong a tone we turn the knob in the opposite direction. Delicate physiological investigations have revealed that the frequency of the neural waves or impulses emitted by a receptor is slow with weak stimulation and fast with strong stimulation. This principle is illustrated very nicely by a series of records published by Graham (6) and reproduced as Figure 3. The frequency of afferent neural impulses is accordingly the code received by the central nervous system which differentiates the various intensities of the same environmental energy. Somehow the central nervous system is evidently able to route nerve currents of different frequencies to the several muscle groups in much the same manner that it does neural impulses coming in over different fiber paths.

CHARACTERISTICS OF THE AFFERENT NEURAL IMPULSE AND ITS PERSEVERATION

It is clear that the immediate determinant of action in organisms is not the stimulating energy, but the neural impulse as finally routed to the muscles. A presumably critical neural determinant intermediate between these two extremes of stimulus (*S*) and response (*R*) is the afferent neural impulse (*s*) at about the time it enters the central ganglia of the nervous system. It is important to note that this afferent impulse (*s*) varies in certain ways not paralleled exactly by changes in the stimulating energy. A particular form of this lack of parallelism is shown clearly in Adrian's graph reproduced as Figure 4 and may be seen more concretely in Figure 3. In all receptors the frequency of receptor discharge begins at a low value and rapidly rises to a comparatively high maximum, after which the rate gradually falls, even though the stimulus continues to act without change.

Certain molar behavioral observations render it extremely probable that the after-effects of receptor stimulation continue to reverberate in the nervous system for a period measurable in seconds, and even minutes, after the termination of the action

types of receptors such as those for light and sound, there is less opportunity for neural interaction than within a given receptor such as the retina. But still the richness of interconnections in the brain furnishes an ample basis for a certain amount of neural interaction here also. This presumption of a tendency for afferent neural impulses to interact before evoking organismic behavior has not yet received detailed physiological proof, and it accordingly has the status of an hypothesis. It will hereafter be called the hypothesis of afferent neural interaction, or, more briefly, the *neural-interaction hypothesis*.

The neural-interaction hypothesis becomes of special importance when it is understood that organisms usually must behave in such a way as to survive in situations which, from the stimulus point of view, are exceedingly complex in the sense of involving the activation of large numbers of receptors at the same time. Moreover, to be adaptive the behavior must sometimes occur only in the presence of a particular combination or configuration of afferent receptor impulses and not in response to any one of the component impulses; sometimes the situation is reversed—the reaction must occur in response to certain component impulses but not to the whole compound; and, finally, the response must sometimes be made to the component impulses irrespective of the occurrence or non-occurrence of other receptor impulses. It is obvious that such a seemingly inconsistent state of the environment presents an extremely difficult problem to the reacting organism, though not necessarily so to the theorist who would explain the degree of success in adaptation which various organisms actually manifest. It will be shown later (p. 349) that the hypothesis of afferent neural interaction when combined with three other behavioral principles will enable us to understand how organisms are able to react to *patterns* or *configurations* of stimulation as such to the extent that this exists in fact. A notable situation of this kind is the response of organisms to distance as received through binocular vision (p. 38 ff.).

THE SPONTANEOUS EMISSION OF IMPULSES BY NERVE CELLS

In this connection we must note another basic neural phenomenon which seems to have rather far-reaching effects upon molar behavior. This is the apparent capacity of individual neurons spontaneously to generate neural impulses, as contrasted with the long-recognized capacity of nerve cells to continue the propagation

of a neural impulse transmitted to them from some other source. Many observations, such as that of the striking variability in organismic behavior under seemingly constant external conditions, have suggested some such tendency. Paul Weiss has demonstrated the phenomenon by means of an extremely clever experiment:

Fragments of spinal cord, including several segments, excised from larval salamanders . . . were grafted into the gelatinous connective tissue of the dorsal fin fold. . . . In seven of the fourteen animals thus operated a limb was grafted at some distance anteriorly or posteriorly to the cord graft. . . . Histological study revealed . . . outgrowth of bundles of nerve fibers into the surroundings. The outgrowing nerve fibers form connections with skin, trunk muscles, and in the presence of a grafted limb, also with the latter. . . . Within a few weeks of the transplantation these isolated cord limb complexes begin to exhibit functional activity, in which three successive phases can be roughly identified. . . . The first phase is characterized by intermittent or almost incessant twitching of the limb muscles. The twitches usually appear in spells, starting with irregular fibrillations and gradually building up to violent convulsions. . . . At the peak of activity, the contractions are remarkably well synchronized, the limb executing strong periodic beats, sometimes at fairly regular intervals of the order of one to several seconds. . . . As a crucial check against the possible intrusion of host innervation . . . the portion of the back containing the grafted units was completely excised and tested in isolation. Even so, the preparations exhibited the same functional activities as before. (9, p. 350-352.)

It is at once evident that the spontaneous firing of nerve cells, if general throughout the nervous systems of normal adult organisms, must when taken in conjunction with the neural-interaction hypothesis imply an incessantly varying modification of both afferent and efferent impulses. From the point of view of the latter it is to be expected that this would produce both qualitative and quantitative variability of reaction to identical environmental receptor stimulation, this variability presumably being a function of the normal "law" of probability. This variability of response is called *oscillation* (p. 304 ff.).

SUMMARY

For most animals, activity is necessary for survival. But not just any movement is sufficient; to be adaptive, movement must be coördinated in a precise manner with the state of various portions of the total environment. This coördination can occur only if the state of the environment is somehow continuously brought to bear

on the motor apparatus. Organic evolution has provided animals with special organs for receiving these physical environmental energies and converting them into neural impulses; with a central mass of neural tissue which, acting as a kind of automatic switch-board, does a fair job of routing the receptor impulses in the direction of the several muscles in adaptive amounts and proportions; and with efferent fibers for transmitting these routed impulses to the individual muscular elements, thus evoking the adaptive behavior.

Receptor responses are prime examples of stimulation—the release of relatively large amounts of resident energy by the action of small amounts of energy from an external source. Reception of touch, temperature, and pain is comparatively simple and requires no comment. Articulatory movement is received by special proprioceptive end organs. Progressive and angular movement is received by the vestibular receptor. Spatial relationships are obtained in various indirect ways, notably through the patterning of impulses received through the ears and eyes. Temporal relationships are also received in various indirect ways but chiefly through the progressive diminution both in the frequency of impulse emission by the receptor during stimulation and in the strength of the “stimulus trace” following the termination of stimulation.

The reactions of organisms are ultimately evoked by the neural impulses which are relayed by the central nervous system to the muscles and glands. In a very real but indirect sense, however, reaction is evoked by the stimulus energies dependent on stimulus objects, though the neural impulse arising from the stimulation of a given receptor is by no means constant, even during the steady action of such a stimulus. The stimulus trace is a hypothetical perseverative process in the receptor areas of the brain which is believed to follow the termination of a receptor discharge with gradually decreasing strength for some seconds and possibly minutes.

Another factor preventing complete agreement or “constancy” between the stimulation element and the afferent discharge to the brain is believed to be neural interaction—the mutual modification of all impulses active in the nervous system at any given time, but especially afferent impulses, more particularly those arising in the same compound receptor organ. This hypothesis makes possible the explanation of many important behavior phenomena, otherwise inexplicable, such as the power of organisms to react to patterns

of stimulation as distinguished from the elements making up the pattern. Finally there must be added the presumptive phenomenon of spontaneous periodic emission of neural impulses by all the cells of the nervous system. This, coupled with the interaction hypothesis, explains many obscure behavior phenomena, notably the variability or oscillation (4, p. 74) of behavior under what may be presumed to be relatively static environmental conditions.

In the light of the preceding considerations, we formulate the following as primary molar behavior principles, postulates, or laws:

POSTULATE 1

When a stimulus energy (S) impinges on a suitable receptor organ, an afferent neural impulse (s) is generated and is propagated along connected fibrous branches of nerve cells in the general direction of the effector organs, via the brain. During the continued action of the stimulus energy (S), this afferent impulse (s), after a short latency, rises quickly to a maximum of intensity, following which it gradually falls to a relatively low value as a simple decay function of the maximum. After the termination of the action of the stimulus energy (S) on the receptor, the afferent impulse (s) continues its activity in the central nervous tissue for some seconds, gradually diminishing to zero as a simple decay function of its value at the time the stimulus energy (S) ceases to act.

POSTULATE 2

All afferent neural impulses (s) active in the nervous system at any given instant, interact with each other in such a way as to change each into something partially different (\bar{s}) in a manner which varies with every concurrent associated afferent impulse or combination of such impulses. Other things equal, the magnitude of the interaction effect of one afferent impulse upon a second is an increasing monotonic function of the magnitude of the first.

NOTES

Pavlov's Statement of the Principle of Afferent Neural Interaction

While Pavlov did not make much systematic use of the principle of afferent neural interaction, he stated it clearly and explicitly, as is shown by the following quotations assembled by Woodbury (10). The italics in all cases are Woodbury's. In connection with the phenomenon of conditioned inhibition, Pavlov remarked: "When the additional stimulus or its fresh trace left in the hemispheres coincides with the action of the positive stimulus, there must result some sort of *special physiological fusion of the effect of the stimuli into one compound excitation partly differing from and partly resembling the positive one.*" (7, p. 70)

Later, when dealing with what in the present work are called *stimulus patterns*

(p. 349 ff.), Pavlov stated: "The cases mentioned above show that a definite interaction takes place between different cells of the cortex, resulting in a fusion or synthesis of their physiological activities on simultaneous excitation. (7, p. 144)

"Plainly the experiments reveal the great importance of the synthesizing activity of the cortical cells which are undergoing excitation. These cells must form, under the conditions of a given experiment, a very complicated excitatory unit, which is functionally identical with the simple excitatory units existing in the case of more elementary conditioned reflexes. *Such active cortical cells must necessarily influence one another and interact with one another*, as has clearly been demonstrated in the case of compound simultaneous stimuli. The mutual interaction between the excited or inhibited cortical elements in the case of compound successive stimuli is more complicated; the effect of an active cortical cell upon the one next excited varies according to the influence to which it was itself subjected by the cell last stimulated. In this way it is seen that the order in which a given group of stimuli taking part in a stimulatory compound are arranged, and the pauses between them are the factors which determine the final result of the stimulation, and therefore most probably the form of the reaction. . . ." (7, pp. 147-148)

While Pavlov clearly recognized the principle which we have called afferent neural interaction as well as the importance of its rôle in the process of conditioning reactions to compound stimuli in distinctive combinations or patterns, it is noteworthy that he did not recognize how the conditioning of reactions to stimulus patterns as such can be derived. In effect this means that he left the process of patterning as a primary principle. It can, however, be derived as a secondary phenomenon from four of his other principles (p. 349 ff.) which appear to be true primary molar laws:

1. Afferent neural interaction (as indicated by the above quotations)
2. Experimental extinction (p. 258 ff.)
3. The generalization of excitation effects (irradiation of excitation, p. 183)
4. The generalization of extinction effects (irradiation of inhibition, p. 262)

Afferent Neural Interaction and the Configuration Psychologies

There is reason to believe that most of the *Gestalt* writers make extensive but implicit use of a principle which is substantially equivalent in some respects at least to the principle of afferent neural interaction. Köhler, however, has been explicit in this respect. In connection with a discussion of perceptual conscious states, he remarks in a recent publication: "Our present knowledge of human perception leaves no doubt as to the general form of any theory which is to do justice to such knowledge: a theory of perception must be a *field theory*. By this we mean that the neural functions and processes with which the perceptual facts are associated in each case are located in a continuous medium; and that the events in one part of this medium influence the events in other regions in a way that depends directly on the properties of both in their relation to each other." (5, p. 55)

Again, in the same general context, Köhler states even more explicitly: "To the extent, therefore, to which these observations bear witness to an interaction which cannot as such be observed within the phenomenal realm, they cannot be understood in purely psychological terms. According to our general program

we shall therefore assume that the interaction occurs among the brain correlates of the perceptual facts in question." (5, p. 63)

Continuing the elaboration of this same general point of view, Köhler adds: "If in a certain sense the correlate of a percept may be said to have a circumscribed local existence we shall none the less postulate that as a dynamic agent it extends into the surrounding tissue, and that by this extension its presence is represented beyond its circumscribed locus. There is no contradiction in these statements. So far as certain properties of the percept process are concerned, this process may be confined within a restricted area, and with this nucleus the percept itself may be associated as an experience. At the same time the presence of such a percept nucleus may lead to further events in its environment, of which we are for the most part not directly aware; but this halo or field of the percept process may be responsible for any influence which the process exerts upon other percept processes." (5, p. 66)

It seems likely that in case an attempt were made to utilize Köhler's interaction principle in developing a thoroughgoing theory of the reaction of organisms to stimulus configurations, it would need to be supplemented by additional principles analogous to those required to supplement Pavlov's parallel principle, and for the same reasons.

REFERENCES

1. ADRIAN, E. D. *The basis of sensation*. New York: W. W. Norton and Co., 1928.
2. FULTON, J. F. *Muscular contraction and the reflex control of movement*. Baltimore: Williams and Wilkins Co., 1926.
3. HULL, C. L. The problem of stimulus equivalence in behavior theory. *Psychol. Rev.*, 1939, 46, 9-30.
4. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
5. KÖHLER, W. *Dynamics in psychology*. New York, Liveright Pub. Co., 1940.
6. MURCHISON, C. *A handbook of general experimental psychology*. Worcester, Mass.: Clark Univ. Press, 1934.
7. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). Oxford Univ. Press, 1927.
8. ROSENBLUTH, A. Central excitation and inhibition in reflex changes of heart rate. *Amer. J. Physiol.*, 1934, 107, 293-304.
9. WEISS, P. Functional properties of isolated spinal cord grafts in larval amphibians. *Proc. Soc. Exper. Biology and Medicine*, 1940, 44, 350-352.
10. WOODBURY, C. B. The learning of stimulus patterns by dogs. *J. Comp. Psychol.* 1943, 35, 29-40..

CHAPTER IV

The Biological Problem of Action and Its Coördination

The receptors of an organism may respond with neural impulses in code corresponding to the near-by presence of food, of an enemy, or of a potential mate. But for the food to be seized and digested, the enemy to be escaped, or the process of reproduction to be initiated, the organism must do something; i.e., it must act. Just as in the last chapter we surveyed the manner in which the processes of organic evolution have solved the receptor problem, so in the present one we shall consider how nature has evolved a solution to the problem of action.

The effector activity of higher organisms is of two major kinds—secretional and motor. Generally speaking, the control of adaptive secretion, such as that of saliva, seems to follow the molar laws of movement. Indeed, some of the most important molar laws of learning were originally isolated by Pavlov and his pupils (2, p. 19 ff.) through the study of conditioned salivary secretions. Because of their greater variety and general interest, discussion in the present chapter will be confined largely to the motor effectors.

THE MOTOR ORGAN

In contrast to the diversity of organs evolved for the reception of environmental energies, the equipment evolved for the execution of movement is comparatively without variety. It consists of only one type of organ—the muscle. There is plenty of variety in the behavior of organisms, but the variety arises mostly from the location and attachments of the several muscles and the permutations and combinations of their joint action rather than from their essential structure. From the point of view of behavioral adaptation, the characteristic function of muscle is contraction. By “contraction” is meant longitudinal shortening which, of course, necessarily means transverse thickening.

The microscopic structure of muscle parallels to a considerable extent its gross structure. The muscle cells are elongated bodies, relatively thick in the middle and tapering at the end. These fibers are activated in their contraction by neural impulses flowing in

from the central nervous system along the fibrous branches of nerve cells. The actual junction of the nerve fibers with the individual

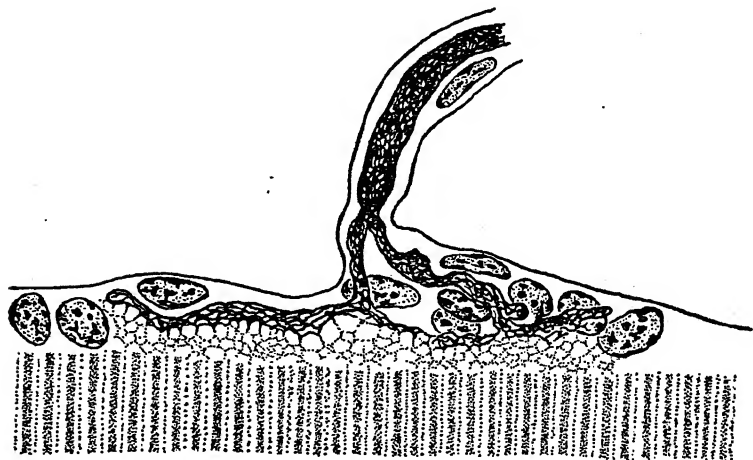


FIG. 6. A section in profile of the motor end-plate of a striated muscle from a young mouse. The vertical striations at the bottom of the figure represent the muscular tissue. (From Fulton, after Boeke, 1, p. 198.)

muscle cell is made by a specialized structure called the neural *end-plate*. Typical connections between nerve fibers and muscles are represented in Figure 6.

THE ALL-OR-NONE LAW OF MUSCLE-FIBER ACTION

Just as the intensity of stimulation needs to be transmitted to the organism by means of a graded neural code, so the rate and general vigor of muscular contraction need to be regulated and controlled in order that adaptation may be adequate. Movement in some situations, such as the flight of a mouse when pursued by a cat, must be rapid and even violent in intensity, whereas in others, such as that of the cat in stalking the mouse, it must be slow and gentle. Nature has evolved a solution to the problem of the gradation of the intensity of action in the "all-or-none" mode of muscle-fiber response to neural discharge.

Within recent years ingenious and delicate physiological investigations have succeeded in isolating small numbers of muscle fibers, in systematically varying the intensity of the neural impulses discharging into them, and in measuring the extent of the resulting

muscular action. The outcome of such an experiment is reproduced as Figure 7. There it may be seen that as the intensity of the neural discharge gradually rises, the amplitude of the reaction of the muscle as a whole increases, *not* gradually but by a series of sharply marked steps; again, as the intensity of the stimulus gradually subsides, the step-wise behavior of the muscle is reversed. Microscopic observation of the individual muscle cells under such experimental conditions confirms the hypothesis that the step-wise rise and fall in the magnitude of muscular activity of

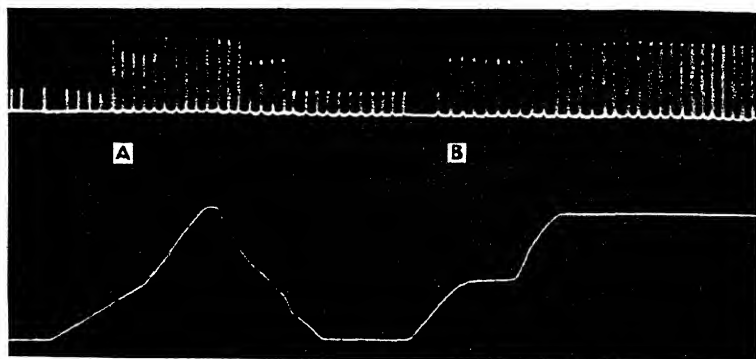


FIG. 7. Reproduction of a photographic record (above) of the movements of a muscle made up of a very small number of fibers. Below is a graphic record of the variation in the magnitude of electrical stimulation (break shocks). Especially note, at the left of the record, that the magnitude of the shock rises and falls gradually yet the muscular reaction rises and falls by a series of discrete steps. These steps are believed to be due to the entrance or cessation of the action of discrete muscle fibers, which is the substance of the all-or-none law, viz., that a particular muscle fiber either responds maximally or not at all, regardless of the amount of stimulation delivered. (From Fulton, *l*, p. 52.)

Figure 7 is due to the fact that at each step in the reaction record a new fiber of the muscle has become active or inactive respectively. Meanwhile those fibers previously innervated by the weaker neural discharge continue also to be innervated by all stronger discharges. The conclusion from this and much other evidence of a similar nature is that each individual muscle fiber has a reaction threshold of its own, below which it will not respond at all and above which it will respond with its maximum contraction. Moreover, the several muscle fibers differ considerably in this reaction threshold, i.e., in the intensity of neural discharge required

UNLEARNED COÖRDINATION OF MUSCULAR ACTIVITY

For some organisms, muscles alone suffice for locomotion and other biological needs. Thus an earthworm is able to crawl about and to secure food; if stimulated by a touch it can withdraw into the safety of its burrow with quite remarkable suddenness. But for the more exacting survival requirements of higher organisms, muscles must be combined with relatively rigid levers. In lower forms of life such as insects, the lever system is on the outside of the body and serves also as a kind of protective armor. The levers of higher organisms consist of bony structures within the body, those levers primarily concerned with locomotion being generally rod-like in form. In order that the bony levers may be moved in various directions, muscles are usually attached to them in pairs called antagonists. Thus the contraction of the biceps on the top of the arm bends or flexes the arm at the elbow, whereas the contraction of the triceps on the opposite side of the arm reverses the movement, straightening or extending the arm. By the combined action of various bones, joints, and muscular contractions, terminal portions of the body such as the hand, say, may be moved in almost any conceivable direction or combination of directions.

The joint action of two or more muscles in the adaptive movement of a portion of an animal's body is called *muscular coördination*. Sometimes a dozen or more distinct muscles are involved in a single coördinated movement, such as the extension of the hind leg of the cat (Figure 8). This coördination (or integration, as it is sometimes called) is brought about in the main by the action of the nervous system. Some of these coördinations are so simple

the survival of the organism in so far as it is dependent upon food. It is noteworthy that one indispensable link in this chain—the flow of the milk—depends upon the very specialized action of the external environment, and once the milk is in the mouth the food itself constitutes the medium which joins the several links of the reflex chain.

SUMMARY

Generally speaking, higher organisms must be more or less active or perish. The effector organs of the typical higher organism consist of glands and muscles. Movement is produced by the longitudinal contraction of the individual fibers making up the muscles. The several fibers of a muscle vary widely in the intensity of the neural impulse necessary to evoke action. For all intensities of neural impulse below the threshold the fiber will be wholly inactive, and for all intensities above the threshold it will be equally and maximally contractile.

The infinitely complex results produced by the simple muscular contractions of organisms are brought about by the various combinations of a relatively small number of muscles, all contracting various degrees and at various rates, several contractions jointly determining the position of a bodily part such as a finger or a foot.

Certain adaptive situations are of such regularity that ready-made chains of reflex receptor-effector connections are adequate for survival, e.g., the blinking of the eyelid at any rough contact with the cornea. In many chains of reflex activity the action of the environment supplies indispensable links of the chain, as in the suckling of a young animal.

REFERENCES

1. FULTON, J. F. *Muscular contraction and the reflex control of movement*. Baltimore: Williams and Wilkins, 1926.
2. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.

DRIVES ARE TYPICAL INTERVENING VARIABLES

It is important to note in this connection that the general concept of drive (D)¹ tends strongly to have the systematic status of an intervening variable or X (see Figure 1), never directly observable. The need of food, ordinarily called hunger, produces a typical primary drive. Like all satisfactory intervening variables, the presence and the amount of the hunger drive are susceptible of a double determination on the basis of correlated events which are themselves directly observable. Specifically, the amount of the food need clearly increases with the number of hours elapsed since the last intake of food; here the amount of hunger drive (D) is a function of observable *antecedent* conditions, i.e., of the need which is measured by the number of hours of food privation. On the other hand, the amount of energy which will be expended by the organism in the securing of food varies largely with the intensity of the hunger drive existent at the time; here the amount of "hunger" is a function of observable events which are its *consequence*. As usual with unobservables, the determination of the exact quantitative functional relationship of the intervening variable to both the antecedent and the consequent conditions presents

¹In case the reader subsequently fails to recall the meaning of this, or any of the other signs employed in the present volume, the significance may be recovered in a moment by consulting the alphabetical list of signs and their meanings given in the Glossary of Symbols (p. 403 ff.).

serious practical difficulties. This probably explains the paradox that despite the almost universal use of the concepts of need and drive, this characteristic functional relationship is not yet determined for any need, though some preliminary work has been done in an attempt to determine it for hunger (1).

INNATE BEHAVIOR TENDENCIES VARY ABOUT A CENTRAL RANGE

With our background of organic evolution we must believe that the behavior of newborn organisms is the result of unlearned, i.e., inherited, neural connections between receptors and effectors (sU_R) which have been selected from fortuitous variations or mutations throughout the long history of the species. Since selection in this process has been on the intensely pragmatic basis of survival in a life-and-death struggle with multitudes of factors in a considerable variety of environments, it is to be expected that the innate or reflex behavior of young organisms will, upon the whole, be reasonably well adapted to the modal stimulating situations in which it occurs.

It may once have been supposed by some students of animal behavior, e.g., by Pavlov and other Russian reflexologists, that innate or reflex behavior is a rigid and unvarying neural connection between a single receptor discharge and the contraction of a particular muscle or muscle group. Whatever may have been the views held in the past, the facts of molar behavior, as well as the general dynamics of behavioral adaptation, now make it very clear not only that inherited behavior tendencies (sU_R) are not strictly uniform and invariable, but that rigidly uniform reflex behavior would not be nearly so effective in terms of survival in a highly variable and unpredictable environment as would a *behavior tendency*. By this expression is meant behavior which will vary over a certain range, the frequency of occurrence at that segment of the range most likely to be adaptive being greatest, and the frequency at those segments of the range least likely to be adaptive being, upon the whole, correspondingly rare. Thus in the expression sU_R , R represents not a single act but a considerable range of more or less alternative reaction potentialities.

The neurophysiological mechanism whereby the type of flexible receptor-effector dynamic relationship could operate is by no means wholly clear, but a number of factors predisposing to variability of reaction are evident. First must be mentioned the spontaneous

impulse discharge of individual nerve cells, discussed above (p. 44). This, in conjunction with the principle of neural interaction operating on efferent neural impulses (*efferent neural interaction*), would produce a certain amount of variability in any reaction. Secondly, the variable proprioceptive stimulation arising from the already varying reaction would, by afferent neural interaction, clearly increase the range of variability in the reaction. Finally, as the primary exciting (drive) stimulus increases in intensity, it is to be expected that the effector impulses will rise above the thresholds of wider and wider ranges of effectors until practically the entire effector system may be activated.

Consider the situation resulting from a foreign object entering the eye. If the object is *very* small the stimulation of its presence may result in little more than a slightly increased frequency of lid closure and a small increase in lachrymal secretion, two effector processes presenting no very conspicuous range of variability except quantitatively. But if the object be relatively large and rough, and if the stimulation continues after the first vigorous blinks and tear secretions have occurred, the muscles of the arm will move the hand to the point of stimulation and a considerable variety of manipulative movements will follow, all more or less likely to contribute to the removal of the acutely stimulating object but none of them *precisely* adapted to that end.

In the case of a healthy human infant, which is hungry or is being pricked by a pin, we have the same general picture, though the details naturally will differ to a certain extent. If the need be acute, the child will scream loudly, opening its mouth very wide and closing its eyes; both legs will kick vigorously in rhythmic alternation, and the arms will flail about in a variety of motions which have, however, a general focus at the mouth and eyes. In cases of severe and somewhat protracted injurious stimulation the back may be arched and practically the entire musculature of the organism may be thrown into more or less violent activity.

to micturate, the need for rest (after protracted exertion), the need for sleep (after protracted wakefulness), and the need for activity (after protracted inaction). The drives concerned with the maintenance of the species are those which lead to sexual intercourse and the need represented by nest building and care of the young.

The primary core or mode of the range of innate or reflex tendencies to action must naturally vary from one need to another if the behavior is to be adaptive. In cases where the rôle of chance as to what movements will be adaptive is relatively small, the behavior tendency may be relatively simple and constant. For example, the acute need for oxygen may normally be satisfied (terminated) by inspiration; the need represented by pressure in the urinary bladder is normally terminated by micturition. It is not accidental that these relatively stereotyped and invariable reactions are apt to concern mainly those portions of the external environment which are highly constant and, especially, the internal environment which is characteristically constant and predictable.

In the case of mechanical tissue injury, withdrawal of the injured part from the point where the injury began is the characteristic reflex form of behavior, and the probability of the effectiveness of such action is obvious. Environmental temperatures considerably below the optimum for the organism tend to evoke shivering and a posture presenting a minimum of surface exposed to heat loss. Temperatures above the optimum tend to produce a general inactivity, a posture yielding a maximum surface for heat radiation, and rapid panting. In certain relatively complex situations such as those associated with the need for food, water, or reproduction, the factor of search is apt to be included as a preliminary. Since extensive search involves locomotion, the preliminary activities arising from these three needs will naturally be much alike.

ORGANIC CONDITIONS WHICH INITIATE THREE TYPICAL PRIMARY DRIVE BEHAVIORS

During recent years physiologists and students of behavior have made important advances in unraveling the more immediate conditions which are associated with the onset of the activities characteristic of the three most complex primary drives—thirst, hunger, and sex. Thirst activities appear from these studies to be initiated by a dryness in the mouth and throat caused by the lack of saliva, which in its turn is caused by the lack of available water in the

FIG. 9. A record of the restless movements of a sleeping student (middle line) and the parallel (hunger) contractions of the student's stomach. Note that the sleeper's restless movements coincide, in general, with the periods of maximal stomach contraction. (After Wada, *?*, p. 29.)

subject's mouth. Then the balloon was inflated and the free end of the tube was attached to a delicate pneumatic recording mechanism. The subjects slept through the night upon an experimental bed which permitted the automatic recording of any restless movements of the sleeper. A presumably typical record so obtained is reproduced as Figure 9. The lower tracing of this record shows at the extreme left the rise of a series of rhythmic contractions of

the stomach, terminating in a kind of cramp followed by a period of cessation. Presently the stomach contractions begin again, and are more or less continuous throughout the remainder of the record. But the main point of this is that the restless movements of the sleeping student (recorded as short vertical oscillations of the middle line in Figure 9) *occurred as a rule only when the stomach contractions were occurring, especially when they were at a maximum.*

Richter (2) attempted to secure parallel records of the stomach contractions and the restless locomotor activity of rats and other organisms to complete the proof of the presumptive relationship afforded by Wada's findings, but was unsuccessful, apparently because of technical difficulties encountered. However, he was able

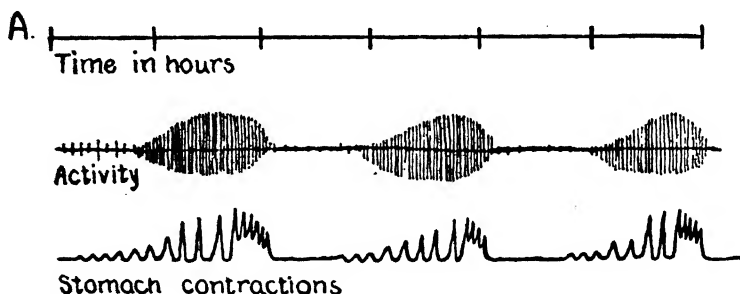


FIG. 10. Diagrammatic representation of the inferred relationship between the periodic stomach contractions of rats and their restless locomotor activity in the living cage. (After Richter, 2, p. 312.)

to show that rats are periodically rather restless in the living cage for a short time before going into a food chamber to eat, after which general activity quickly subsides. The periodicity of these restless movements is about the same as that known to occur with the stomach contractions. Richter accordingly concludes from a convincing array of such indirect evidence that the relationship between random, restless activity and the gastric hunger contractions is substantially that shown in Figure 10. It is to be observed that this figure is not a record but, rather, a diagrammatic representation of an inferential relationship. Nevertheless, Richter's reasoning is fairly convincing and Figure 10 quite probably represents the true situation.

The functional interpretation of this restless behavior is that an organism which moves about more or less continuously will in

general traverse a wide area and consequently will be more likely to encounter food than if it remains quietly in one place.

TYPICAL STUDIES OF SEXUALLY MOTIVATED ACTIVITY

Richter has also shown that the male rat displays much more restless locomotor activity when the sex drive is operating than when it is not. He placed male rats in drum-like cages pivoted on a central axis in such a way that if the animal attempted to climb the circular side of the cage its weight would turn the drum. Automatic counting devices aggregated the amount of this kind of

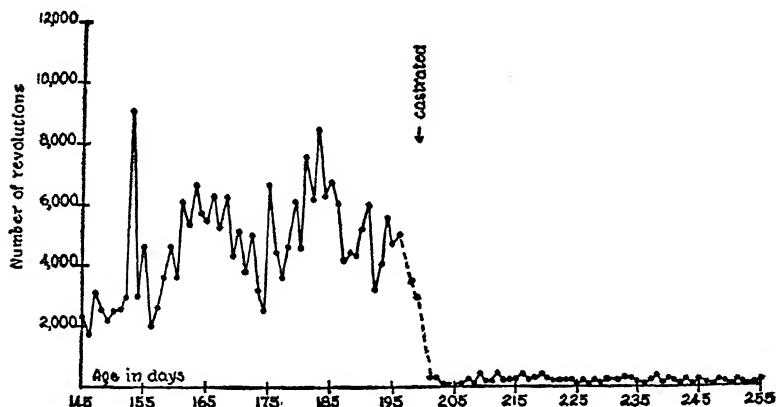


FIG. 11. Graphic representation of restless locomotor activity of a male albino rat in a revolving cage before and after castration. (After Richter, *2*, 329.)

locomotor activity by days. A graphic representation of the numerical values so obtained is shown at the left of Figure 11. At about the 195th day the rat was castrated. Note the abrupt drop in the restless locomotor activity. Even if one makes a certain allowance for the shock produced by the operation as such, the inference is that when the hormone secreted by the testes is in the blood, the animal is generally active, but when this hormone is withdrawn through castration, generalized locomotor activity falls to a relatively low level and remains there.

Wang (4) has shown by analogous means that the female rat is maximally active in this same restless fashion about every fourth day, the two or three days between showing a relatively small amount of activity. A considerable number of cycles from a single

female rat are represented graphically in Figure 12 (2). That these maxima of locomotor activity are coincident with periodically recurring sex drive is shown by the fact that on the occasion of the maxima such animals are receptive to the sexual advances of the male.

The functional interpretation of these studies is similar to that

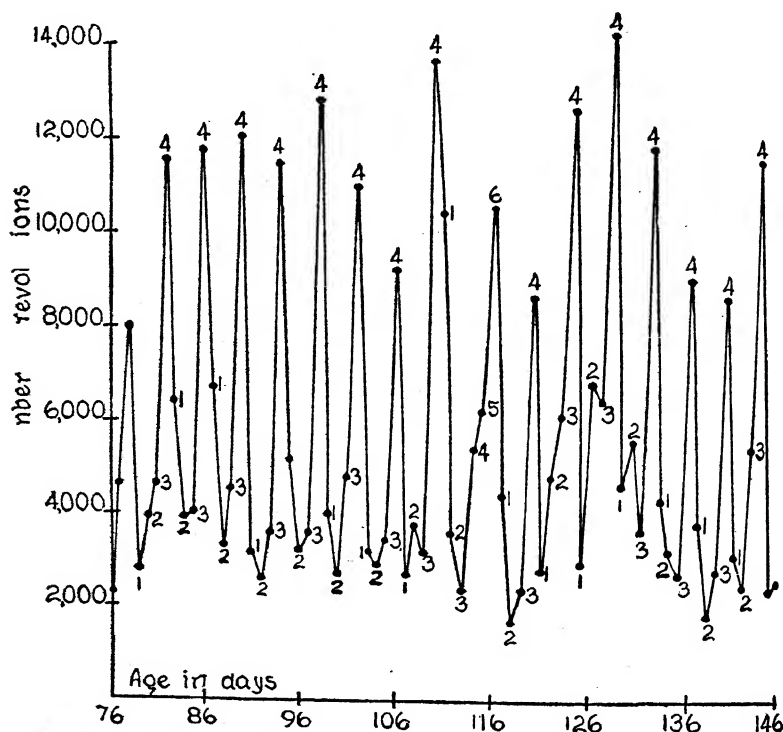


FIG. 12. Graphic representation of the locomotor activity of a female albino rat over a series of days. Note the prevalence of a four-day cycle of activity. On the days of maximum activity these animals are sexually receptive. (After Richter, 2, 321.)

of the investigations involving hunger; an organism which moves about continuously will traverse a wide area and consequently will be more likely to encounter a mate than will an organism which remains in a single place.

SUMMARY

Animals may almost be regarded as aggregations of needs. The function of the effector apparatus is to mediate the satiation of

these needs. They arise through progressive changes within the organism or through the injurious impact of the external environment. The function of one group of receptors (the drive receptors) is to transmit to the motor apparatus, via the brain, activating impulses corresponding to the nature and intensity of the need as it arises. Probably through the action of these drive receptors and receptor-effector connections preëstablished by the processes of organic evolution, the various needs evoke actions which increase in intensity and variety as the need becomes more acute.

Because of the inherently fortuitous nature of the environmental circumstances surrounding an organism when a state of need arises, the kind of behavior which will be required to alleviate the need is apt to be highly varied. For this reason rigid receptor-effector connections could not be very effective in terms of organismic survival. Accordingly we find, as a matter of fact, that innate molar response to a given state of need presents a considerable range of activity, the activity often consisting of a sequence of short cycles of somewhat similar yet more or less varied movements. Such behavior cycles are believed normally to show a frequency distribution in which those acts most likely to relieve the need occur most often, and those acts less likely to terminate it occur correspondingly less often. Thus reflex organization (sU_R) has more than one string to its bow; if one reaction cycle does not terminate the need, another may. The modal form of reaction will also be strongest, so that it will usually occur not only most frequently, but earliest and probably will remedy the situation; but if the environment chances to be such that some other simple action sequence is required, in due course this action sequence will probably occur and the organism will survive. Finally, in still more complicated situations a particular combination of these acts may terminate the need. In this way innate behavior tendencies are organized on a genuine but primitive trial-and-error basis.

Lastly, it is to be noted that just as food-seeking activity begins long before the organism is in acute need of food, so other drives become active long before heat or cold or pain becomes seriously injurious or even in the least harmful. In short, it may be said that drives become active in situations which, if more intense or if prolonged, *would* become injurious. Once more, then, the probability aspect of primitive behavior tendencies becomes manifest.

In the light of the preceding considerations we formulate the following primary molar behavior principle:

POSTULATE 3

Organisms at birth possess receptor effector connections (sUR) which, under combined stimulation (S) and drive (D), have the potentiality of evoking a hierarchy of responses that either individually or in combination are more likely to terminate the need than would be a random selection from the reaction potentials resulting from other stimulus and drive combinations.

NOTES

The Rôle of Adaptation in Systematic Behavior Theory

The emphasis in this and preceding chapters on the general significance of organic evolution in adapting organisms to meet critical biological emergencies calls for a word of comment, lest the reader be misled in regard to the rôle that adaptation, as such, plays in the present system. It is the view of the author that adaptive considerations are useful in making a preliminary survey in the search for postulates, but that once the postulates have been selected they must stand on their own feet. This means that once chosen, postulates or principles of behavior must be able to yield deductions in agreement with observed detailed phenomena of behavior; and, failing this, that no amount of *a priori* general adaptive plausibility will save such a postulate from being abandoned.

Problems Associated with the Use of Drive (D) as an Intervening Variable

Most writers on behavior theory utilize the concept of need or some equivalent such as drive, though hardly one of them has faced squarely the associated problem of finding the two equations necessarily involved if the concept of drive is to take its place in a strict mathematical theory of behavior (5). In the case of hunger, for example, there must be an equation expressing the degree of drive or motivation as a function of the number of hours' food privation, say, and there must be a second equation expressing the vigor of organismic action as a function of the degree of drive (D) or motivation, combined in some manner with habit strength. A correlated task of some magnitude is that of objectively defining a unit in which to express the degree of such a motivational intervening variable (see p. 226 ff.).

Now it is a relatively easy matter to find a single empirical equation expressing vigor of reaction as a function of the number of hours' food privation or the strength of an electric shock, but it is an exceedingly difficult task to break such an equation up into the two really meaningful component equations involving hunger drive (D) or motivation as an intervening variable. It may confidently be predicted that many writers with a positivistic or anti-theoretical inclination will reject such a procedure as both futile and unsound. From the point of view of systematic theory such a procedure, if successful, would present an immense economy. This statement is made on the assumption that motivation (D) as such, whether its origin be food privation, electric shock, or whatever, bears a

certain constant relationship to action intensity in combination with other factors, such as habit strength. If this fundamental relationship could be determined once and for all, the necessity for its determination for each special drive could not then exist, and so much useless labor would be avoided. Unfortunately it may turn out that what we now call drive and motivation will prove to be so heterogeneous that no single equation can represent the motivational potentiality of any two needs. Whether or not this is the case can be determined only by trial.

REFERENCES

1. PERIN, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. *J. Exper. Psychol.*, 1942, 30, 93-113.
2. RICHTER, C. P. Animal behavior and internal drives. *Quar. Rev. Biology*, 1927, 2, 307-343.
3. WADA, T. An experimental study of hunger in its relation to activity. *Arch. Psychol.*, 1922, No. 57.
4. WANG, G. H. The relation between "spontaneous" activity and oestrous cycle in the white rat. *Comp. Psychol. Monog.*, 1923, No. 6.
5. YOUNG, P. T. *Motivation of behavior*. New York: John Wiley and Sons, 1936.

CHAPTER VI

The Acquisition of Receptor-Effector Connections— Primary Reinforcement

We have seen above that organisms require a considerable variety of optimal conditions if the individual and the species are to survive. In many cases where the conditions, particularly internal ones, deviate materially from the optimum, complex automatic physiological processes make the adjustment. An example of this is the remarkable manner in which the blood is maintained at a practically constant state in the face of a great variety of adverse conditions. This type of automaticity has been called by Cannon the "wisdom of the body" (2). In the case of certain other needs, and here lies our chief interest, the situation is remediable only by movement, i.e., muscular activity, on the part of the organism concerned. The processes of organic evolution have produced a form of nervous system in the higher organisms which, under the conditions of the several needs of this type, will evoke without previous learning a considerable variety of movements each of which has a certain probability of terminating the need. This kind of activity we call *behavior*.

THE PROBLEM AND GENERAL NATURE OF LEARNING

It is evident, however, that such an arrangement of ready-made (inherited) receptor-effector tendencies, even when those evoked by each state of need are distinctly varied, will hardly be optimally effective for the survival of organisms living in a complex, highly variable, and consequently unpredictable environment. For the optimal probability of survival of such organisms, inherited behavior tendencies must be supplemented by learning. That learning does in fact greatly improve the adaptive quality of the behavior of higher organisms is attested by the most casual observation. But the detailed nature of the learning process is not revealed by casual observation; this becomes evident only through the study of many carefully designed and executed experiments.

The essential nature of the learning process may, however, be stated quite simply. Just as the inherited equipment of reaction

tendencies consists of receptor-effector connections, so the process of learning consists in the strengthening of certain of these connections as contrasted with others, or in the setting up of quite new connections. In many ways this is the highest and most significant phenomenon produced by the processes of organic evolution. It will be our fascinating task in the present and several succeeding chapters to tease out bit by bit from the results of very many experiments the more important molar laws or rules according to which this supremely important biological process takes place.

In accordance with the objective approach outlined in Chapter II we must regard the processes of learning as wholly automatic. By this it is meant that the learning must result from the mere interaction between the organism, including its equipment of action tendencies at the moment, and its environment, internal as well as external. Moreover, the molar laws or rules according to which this interaction results in the formation or strengthening of receptor-effector connections must be capable of clear and explicit statement. Recourse cannot be had to any monitor, entelechy, mind, or spirit hidden within the organism who will tell the nervous system which receptor-effector connection to strengthen or which receptor-effector combination to connect *de novo*. Such a procedure, however it may be disguised, merely raises the question of the rule according to which the entelechy or spirit itself operates; this, of course, is the original question all over again and clarifies nothing.

THE STRENGTHENING OF INNATE RECEPTOR-EFFECTOR CONNECTIONS

Because of its presumptive temporal priority in the life of the organism, we shall consider first the problem of the selective strengthening of one among a variety of inherited movement tendencies evoked by a need in a particular environing situation. This can perhaps best be done by means of an illustrative experiment, even though some of the reaction tendencies there operative may already have been modified by learning. The experimental procedure and the results will be described in a little detail, out of consideration for readers who have slight knowledge of the routine methodologies characteristic of behavior laboratories.

Demonstration Experiment A. The laboratory in which the experiment is performed is without windows and its walls are painted black; this gives the room an appearance of being rather dimly illuminated, though in fact it is not. On a table rests a black wooden apparatus about two feet long, a foot wide, and a foot high. It has a hinged glass lid which permits clear observation of the interior. The floor of the box consists of small transverse rods of stainless steel placed about a quarter inch apart. Midway between the two ends of the box is a partition consisting of the same type of metal rods similarly arranged but placed vertically. This partition or barrier reaches to within about four inches of the lid. A two-throw electric switch permits the charging of the floor rods of either compartment and of the partition with a weak alternating current.

On a second table nearby there rests a wire cage containing a sleek and lively albino rat about one hundred days of age. The laboratory technician opens the lid of the cage and the rat at once stands up on its hind legs with its head and forepaws outside the aperture. The technician grasps the rat about the middle with his bare hand and transfers it to one of the compartments of the apparatus. The animal, after a brief pause, begins moving about the compartment, sniffing and inspecting the various parts, often stretching up on its hind legs to its full length against the walls of the box.

After some minutes the technician throws the switch which charges both the partition and the grid upon which the rat is standing. The animal's behavior changes at once; in place of the deliberate exploratory movements it now displays an exaggeratedly mincing mode of locomotion about the compartment interspersed with occasional slight squeaks, biting of the bars which are shocking its feet, defecation, urination, and leaps up the walls. These reactions are repeated in various orders and in various parts of the compartment; sometimes the same act occurs several times in succession, sometimes not. After five or six minutes of this variable behavior one of the leaps carries the animal over the barrier upon the uncharged grid of the second compartment. Here after an interval of quiescence and heavy breathing the animal cautiously resumes exploratory behavior, much as in the first compartment. Ten minutes after the first leap of the barrier the second grid is charged and the animal goes through substantially the same type of variable behavior as before. This finally results in a second leaping of the barrier and ten minutes more of safety, after which this grid is again charged, and so on. In this way the animal is given fifteen trials, each terminated by a leap over the barrier.

A comparison of the animal's behavior leading to his successive escapes from the charged grid shows clear evidence of learning in that upon the whole the time from the onset of the shock to the escape became progressively less, until at the last few trials the leaping reaction followed the onset of the shock almost instantaneously. Meanwhile the competing reactions gradually decreased in number until at the end they ceased to occur altogether. Once or

twice the rat even leaped the barrier before the shock was turned on at all. Here, then, we have a clear case of *selective* learning.

It is evident from the foregoing that the final successful competition of the reaction of leaping the barrier (R_4) with the various futile reactions of the series such as leaping against the wooden walls of the apparatus (R_1), squeaking (R_2), and biting the floor bars (R_3) must have resulted, in part at least, from a differential strengthening of R_4 . It is also evident that each of these competing reactions was originally evoked by the slightly injurious effects of the current on the animal's feet (the condition of need or drive, D) in conjunction with the stimulation (visual, cutaneous, etc.) arising from the apparatus at about the time that the reaction took place. The stimulation arising from the apparatus at the time of the respective reactions needs to be designated specifically: leaping against the wall will be represented by S_A ; squeaking, by S_A' ; biting, by S_A'' ; and leaping the barrier, by S_A''' . It is assumed that preceding the learning, the leaping of the barrier was evoked by a compound connection between the receptor discharges s_D and s_A , arising from S_D and S_A respectively, and R_4 ; i.e., R_4 must have been evoked jointly by the converging connections, $s_D \longrightarrow R_4$ and $s_A \longrightarrow R_4$. These are the connections which evidently have been strengthened or reinforced. Because of this, learning is said to be a process of *reinforcement*.

We must now approach the central problem of learning by attempting to formulate the rule according to which primary reinforcement occurred in this case of selective learning. More specifically, we must ask the rule according to which the connections $s_D \longrightarrow R_4$ and $s_A''' \longrightarrow R_4$ were differentially strengthened so as to become dominant over the numerous other reaction tendencies. The most plausible statement of this rule at present available is: *Whenever a reaction (R) takes place in temporal contiguity with an afferent receptor impulse (\dot{s}) resulting from the impact upon a receptor of a stimulus energy (\dot{S}), and this conjunction is followed closely by the diminution in a need (and the associated diminution in the drive, D , and in the drive receptor discharge, s_D), there will result an increment, $\Delta(\dot{s} \longrightarrow R)$, in the tendency for that stimulus on subsequent occasions to evoke that reaction.* This is the "law" of *primary reinforcement*.¹

Thus in the case of learning exhibited in Demonstration Ex-

¹ Actually, of course, this formulation has only the status of an hypothesis. The term *law* is here used in much the same loose way that Thorndike has

periment A, both of the afferent stimulus impulses, s_D and s_A''' , were obviously active when reaction R_4 occurred because they evoked it. Moreover, this conjunction of s_D and s_A''' with R_4 was followed immediately by the termination of the shock effects or need, and so by a reduction in s_D . But by the principle of primary reinforcement just formulated this reduction in the need and drive

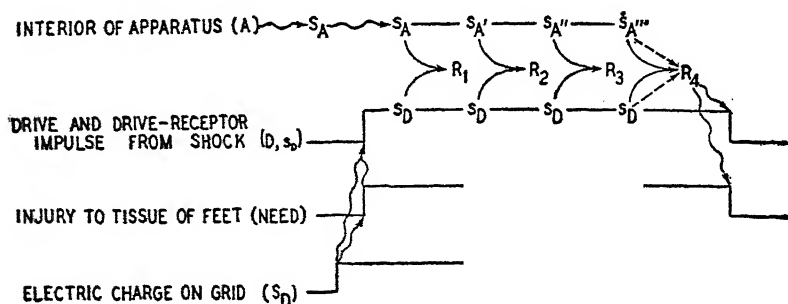


FIG. 13. Diagrammatic representation of the process of strengthening or reinforcing the connections between $s_D \rightarrow r_4$ and $s_A''' \rightarrow r$. The step-like rises and falls of the several horizontal lines such as those of D and s_D , the shock to the tissue and S_D , represent the rise from zero and the fall of the respective processes. The arrows with wavy shafts ($\sim \rightarrow$) represent a physical causal relationship other than by way of receptor-effector stimulus evocation. Thus the rise of the current on the grid (S_D) causes the shock to the tissue of the animal's feet, the response of the receptor in the skin (s_D) of those regions, and the drive (D) or motivation to action. The separation of the foot from the grid by the act of jumping (R_4) terminates simultaneously the injurious action or need, the receptor discharge (s_D), and the drive (D), though the current on the grid remains unchanged. It is the reduction in the drive receptor impulse (s_D) and the drive (D) which are believed to be the critical factors in the process of reinforcement. The arrows with solid shafts (\longrightarrow), whether curved or straight, separate or jointed, represent receptor-effector relationships in existence before the learning process here represented occurred. The arrows with broken shafts ($---\rightarrow$) represent receptor-effector connections here in process of formation. Distance from left to right represents the passage of time.

(D) will, through the associated decrement in the drive receptor impulse (s_D), result in increments, $\Delta(s_A''' \rightarrow R_4)$ and $\Delta(s_D \rightarrow R_4)$, to the tendency for such conjoined afferent stimulus elements (s_A''' and s_D) to evoke the reaction (R_4) on subsequent occasions. The major dynamic factors of this process are represented diagrammatically in Figure 13.

used it in his famous expression, "law of effect," to which the above formulation is closely related (9, p. 176).

THE ACQUISITION OF NEW RECEPTOR-EFFECTOR CONNECTIONS

We proceed now to the consideration of the formation of a genuinely new receptor-effector connection. This turns out to be only a special case of the law of primary reinforcement which we have just formulated. Moreover, this type of selective learning may be demonstrated by means of an experiment differing only slightly from the one already considered at some length.

Demonstration Experiment B. The variation of Demonstration Experiment A consists merely in the sounding of a buzzer continuously from a time two seconds before the shock is turned on the grid until the animal leaps the barrier. The course of the learning is much as in the preceding experiment up to the point where the animal has eliminated all of the original acts except that of leaping the barrier. At this point, however, *the animal begins occasionally to leap over the barrier during the first two seconds of the sounding of the buzzer.*

It will be evident at once that this outcome follows directly from the law of primary reinforcement stated above because, as

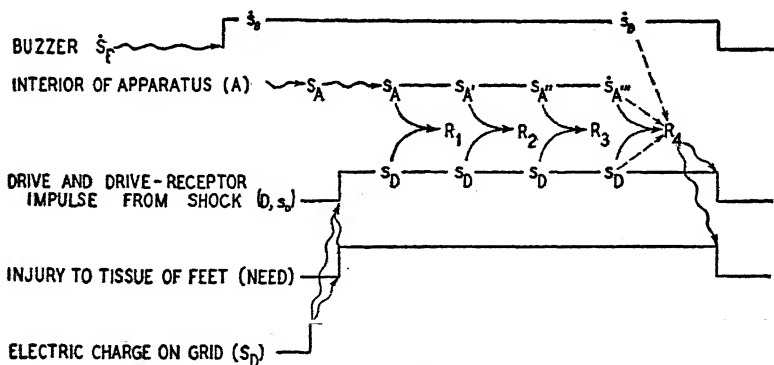


FIG. 14. Diagrammatic representation of the dynamic factors involved in the setting up of a new receptor-effector connection in a selective learning situation, Demonstration Experiment B. With the exception of the upper line representing the rise and continuation of the buzzer stimulus and its receptor discharge, and the connection $\dot{s}_B \dashrightarrow R_4$ in process of formation, this diagram is exactly the same as that of Figure 13.

shown by Figure 14, the receptor discharge (\dot{s}_B) resulting from the action of the buzzer vibrations (\dot{s}_B) on the mechanisms of the internal ear has a conjunction (just as have s_D and s_A''') with R_4 ; and the termination of the need, and that of the associated drive (D) and drive receptor discharge (s_D), constitute a reinforcing

state of affairs exactly as in the first form of the experiment. Thus after reinforcement in the manner described there exists in the body of the organism a habit represented diagrammatically by the broken-shafted arrows of Figures 14 and 15.

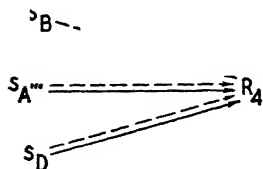


FIG. 15. Diagram representing the results of a reinforcement in which a quite new receptor-effector connection ($S_B \dashrightarrow R_4$) has been set up as shown in Figure 14. The arrows with double shafts represent the combination of the old and the newly acquired receptor-effector connections between S_A''' and S_D , respectively, and R_4 . The unbroken shafts represent either unlearned receptor-effector connections or at least connections in existence before the particular learning under consideration began.

From other experiments (p. 209) it is known that the receptor-effector connections from several stimulus aggregates, all converging simultaneously upon the same reaction, tend strongly to summate (see p. 223 ff.). As a result the action of all three connections would be stronger than that of the entire group less any one. Because of the absence of $S_D \longrightarrow S_D \dashrightarrow R_4$, during the uncharged state of the grid, it is to be expected that the rat would at first wait until the delivery of the shock before jumping. However, with additional reinforcements the action-evocation strengths of S_B and S_A''' finally become great enough when combined to evoke the reaction before the shock is delivered. Because this reaction is the same as that which usually occurs only after the onset of the shock, it is called *antedating* or

anticipatory; since it results in total escape from the injury produced by the shock, it is highly adaptive.

Finally, with still more reinforcements, the receptor-effector connections become so strong that the relatively static connection, $S_A''' \longrightarrow S_A''' \dashrightarrow R_4$, alone will evoke the jumping reaction, i.e., the stimulus of the apparatus alone will evoke the adaptive response, before the onset of either the buzzer or the shock.

THE CONDITIONED REFLEX

A special case of the action of the principle of reinforcement sketched above is found in the type of experiment in which there is set up what is indifferently called the *conditioned reflex* or the *conditioned reaction*. Despite a certain artificiality, the relative simplicity of this type of experiment has permitted the isolation of a large number of molar laws, particularly in the laboratory of

I. P. Pavlov, the great Russian physiologist (8). The typical conditioned-reflex experiment is conducted in such a way as (1) to set up new receptor-effector connections rather than, as in Demonstration Experiment A, to strengthen connections already strong enough in combination to evoke overt reactions, and (2) to eliminate the necessity of selecting one reaction from the numerous varied reactions normally evoked by the conjunction of a need in a stimulating situation. The elimination of the complication characteristic of selective learning is brought about by the simple expedient of reinforcing the first, i.e., the dominant, reaction of the potential sequence of competing tendencies normally evoked by a need situation. Since the process of primary reinforcement employed in such experiments usually involves the termination of the need, the duration of the need is so brief that the weaker members of the potential action group rarely become overt.

In order to make the distinctive characteristics of the conditioned-reflex experiment especially clear, the following example of conditioned-reflex learning was designed in such a way as to be an exact parallel to Demonstration Experiment A.

Demonstration Experiment C. A dog is habituated to stand in a stock or wooden framework resting on a laboratory table. A soft leather moccasin containing an exposed electric grid in its sole is laced to the dog's foot. The moccasin is attached to a light hinged board which is held down by a coil spring. The electric circuit which conducts the alternating current to the grid passes across a connection which is broken when the dog's footboard is lifted one inch at the point of moccasin attachment.

The experiment is conducted as follows: A buzzer is sounded two seconds before a shock is delivered to the dog's foot through the moccasin grid. The resulting shock produces as its dominant reaction a reflex lifting of the shocked foot which breaks the circuit, thus terminating the shock. No doubt the dog makes many other muscular contractions in addition to those which result in the lifting of the foot, but these are usually neglected in such experiments; the main point is that the foot-lifting act always does take place at once. After ten minutes the buzzer-shock combination is repeated with the same results as before. This is continued until fifteen reinforcements have taken place.

By the principle of primary reinforcement outlined above, the receptor discharge (\dot{s}_c)¹ arising from the buzzer vibrations (the "conditioned stimulus" or \dot{S}_c) is temporally conjoined with the foot-

¹ The symbols \dot{s}_c , s_u , and R_u have become conventionalized in the conditioned-reflex literature and are here used with their conventional meaning. The dot has been placed above the s in \dot{s}_c in order also to conform to the usage of the present work.

lifting reaction (the so-called "unconditioned reaction" or R_u), and this conjunction is followed at once by the termination of the shock or need and of the associated drive receptor impulse (s_u) which constitutes the reinforcing state of affairs. As a result there must be set up an increment to the associative connection between the afferent receptor impulse produced by the buzzer vibrations and the foot-lifting reaction. Thus after a sufficient number of repetitions there must arise the new superthreshold receptor-effector connection, $\dot{s}_c \rightarrow R_u$, and the dog begins regularly to lift his foot promptly at the sound of the buzzer.

Quite naturally, exactly as in the preceding experiments, there is also set up a connection between the various static stimuli arising from the apparatus (\dot{S}_A) and the reaction (R_u) because the former also are active in conjunction with the foot-lifting act and so become connected along with the so-called conditioned stimulus, thus: $\dot{S}_A \rightarrow R_u$. As a result of this connection the dog will frequently lift his foot when the buzzer is not sounding, just as the rat would sometimes leap the barrier when neither the buzzer nor the shock was acting. In the case of the dog this unadaptive behavior is discouraged by the spring which tends to hold down the footboard.

THE CONDITIONED REFLEX A SPECIAL CASE OF ORDINARY LEARNING REINFORCEMENT

Demonstration Experiment C presents a fairly typical example of conditioned-reflex learning, though it is characteristic of the school of Bechterev (1) rather than that of Pavlov.¹ We have already seen that the acquisition of a quite new receptor-effector connection, a phenomenon of conditioned-reflex learning, is deducible from the conditions of Demonstration Experiment C on the basis of the law of primary reinforcement formulated above in connection with a typical bit of selective learning (Demonstration Experiment A). Because of the current differences of opinion concerning the relationship between selective learning and conditioned-reflex learning, an explicit and somewhat detailed comparison of them as types will now be made. In order to facilitate such a

¹The salivary conditioned-reflex technique of Pavlov involves certain complexities, the consideration of which must be delayed until the next chapter when the necessary groundwork for their adequate understanding will have been laid.

comparison, Figure 16 has been constructed to represent in considerable detail the dynamic factors here conceived to be involved in conditioned-reflex learning, in close parallel with the representa-

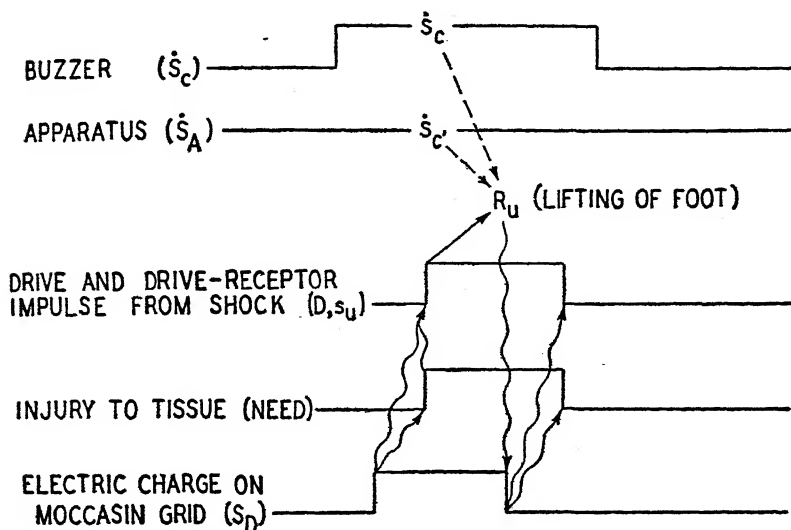


FIG. 16. The conditioned-reflex learning of Demonstration Experiment C represented according to the primary reinforcement formulation presented in the text. The R_u terminates the shock or drive (S_D) which in turn terminates both the tissue injury and the associated receptor impulses. Presumably the latter is the essential constituent of the reinforcing state of affairs which sets up the connections $\dot{S}_c \dashrightarrow R_u$ and $\dot{S}_A \dashrightarrow R_u$.

tion of the process of selective reinforcement of Demonstration Experiment A as presented in Figure 13.

A comparative study of Figures 13 and 16 verifies explicitly what has already been pointed out. There it may be seen that:

1. Simple selective learning (Figure 13) involves the selection of a particular reaction from numerous alternative reactions which are evoked more or less at random by the need and the stimulus situation jointly, whereas in conditioned-reaction learning (Figure 16) there is only one (and the same) conspicuous act involved at each reinforcement trial.

2. Simple selective learning may involve the mere strengthening of receptor-effector connections, already of superthreshold strength before the beginning of the experiment, whereas in conditioned-reflex learning there results, typically, a completely new receptor-effector connection.

From this point of view, Demonstration Experiment B offers a kind of transition from the extremes of Demonstration Experi-

ments A to C, since it is clearly a case of selective learning yet it involves the setting up of a receptor-effector connection *de novo* and, at the same time, the presumptive strengthening of other connections already superthreshold in strength. It must be added that conditioned-reaction experiments may also be arranged in such a way as to strengthen superthreshold connections and that the terminal phases of conditioned-reaction learning inevitably involve the strengthening of connections set up *de novo* in the early stages of a given learning process.

These last considerations suggest that the differences between the two forms of learning are superficial in nature; i.e., that they do not involve the action of fundamentally different principles or laws, but only differences in the *conditions* under which the principle operates (6, Theorem I). This preliminary impression is confirmed when we make a comparison of the two situations (as represented by Figures 13 and 16). On one critical point both cases are identical—the reinforcing state of affairs in each consists in the abolition of the shock injury or need, together with the associated decrement in the drive and drive receptor impulse, at once after the temporal conjunction of the afferent receptor discharge and the reaction. This is, of course, all in exact conformity with the law of primary reinforcement formulated above (p. 71).

$\dot{S}_c \longrightarrow R_u$

FIG. 17. Conventional diagram of the dynamics of conditioned-reflex learning common in American textbooks of elementary psychology. The circle surrounding R_c is to indicate that the preexperimental reaction of \dot{S}_c is inconspicuous, unknown, or non-existent.

We pass now to the consideration of an alternative interpretation of the conditioned reflex, namely, that held by Pavlov (8), its greatest exponent. This is represented fairly well by Figure 17, a diagram frequently employed to illustrate Pavlov's views by American writers of elementary textbooks (3, p. 381; 7, p. 245). As this diagram suggests, Pavlov agrees substantially with the "law

of reinforcement" formulated above, as well as with the "law of effect" as formulated by Thorndike, in holding that the conditioned stimulus S_c (or its trace, \dot{s}_c) must have approximate temporal conjunction with the unconditioned reaction (R_u) before the receptor-effector connection can be established. Pavlov differs from the law of reinforcement by regarding as the critical element of the reinforcing state of affairs the occurrence of S_u , in this case the *onset*

of the shock. On the other hand, the critical element in the reinforcing state of affairs by our own hypothesis is the reduction in the drive receptor impulse (s_D or s_u) which accompanies the reduction of the need, i.e., reduction of the physiological injury to the tissue of the feet, caused by the termination of the shock.

It is an easy matter to show the inadequacy of Pavlov's formulation as a general theory of learning by applying it to the case of simple selective learning presented by Demonstration Experiment A (Figure 13). According to Pavlov's hypothesis, every one of the false reactions R_1 , R_2 , R_3 , etc., should have been reinforced just as should R_4 , because (in the conditioned-reflex terminology) each is evoked by the unconditioned stimulus (S_u). In that case if any selection at all could be expected to occur that reaction which takes place the most frequently would be the one selected rather than the reaction which actually would set in motion a causal sequence leading to the termination of the need. Yet innumerable experiments show that, other things equal, the reaction which is followed at once by a diminution in a primary need will be selected, regardless of its original frequency of occurrence.

It is not difficult to understand how Pavlov could have made such an error. His mistaken induction was presumably due in part to the exceedingly limited type of experiment which he employed. Within the range of his restricted procedures his formulation was consistent with all of the facts and observed relationships. Had he worked even a little with simple selective learning he would doubtless have seen his error and corrected it. Actually he seems not to have occupied himself greatly with the problem of the exact nature of the reinforcing state of affairs; he was interested mainly in discovering the detailed characteristics of conditioned-reflex phenomena by an intensive experimental attack. In this he was eminently successful.

SUMMARY

The infinitely varied and unpredictable situations of need in which the higher organisms find themselves make any form of ready-made receptor-effector connections inadequate for optimal probability of survival. This natural defect of inherited reaction tendencies, however varied, is remedied by learning. Learning turns out upon analysis to be either a case of the differential strengthening of one from a number of more or less distinct reac-

tions evoked by a situation of need, or the formation of receptor-effector connections *de novo*; the first occurs typically in simple selective learning and the second, in conditioned-reflex learning. A mixed case is found in which new receptor-effector connections are set up at the same time that selective learning is taking place.

An inductive comparison of these superficially rather divergent forms of learning shows one common principle running through them all. This we shall call the *law of primary reinforcement*. It is as follows: *Whenever an effector activity occurs in temporal contiguity with the afferent impulse, or the perseverative trace of such an impulse, resulting from the impact of a stimulus energy upon a receptor, and this conjunction is closely associated in time with the diminution in the receptor discharge characteristic of a need, there will result an increment to the tendency for that stimulus on subsequent occasions to evoke that reaction.* From this principle it is possible to derive both the differential receptor-effector strengthening of simple selective learning and the acquisition of quite new receptor-effector connections, characteristic of conditioned-reflex learning as well as of certain forms of selective learning. Pavlov puts forward the alternative hypothesis that the critical element in the reinforcing state of affairs is the occurrence of the unconditioned stimulus. This formulation fits conditioned-reflex phenomena but breaks down when applied to selective learning situations, a fact which shows it to be an inadequate inductive generalization. Fortunately the inadequacy of this interpretational detail of Pavlov's work in no way detracts from the scientific value of the great mass of empirical findings produced by his laboratory.

NOTES

Is the Reinforcing State of Affairs in Learning Necessarily the "Effect" of the Act Being Reinforced?

It has already been suggested that the hypothesis as to the reinforcing state of affairs adopted in the present work is distinctly related to that of Thorndike's "law of effect." Thorndike seems to have coined this expression because the state of affairs which has been found empirically to be necessary in order to produce differential reinforcements, as in Demonstration Experiments A, B, and C, under ordinary circumstances comes literally as the effect of the reaction which is reinforced. This cause-and-effect relationship is shown explicitly by means of the wavy-shafted arrows leading from the act reinforced, e.g., R_4 in Figures 13 and 14, which patently causes the termination of the injurious action to the foot tissue and of the receptor impulses, s_D . A strictly parallel though slightly more complex causal relationship is seen in Figure 16, where R_{11} terminates the current

on the grid by interrupting the circuit and this, in turn, terminates the shock to the foot and at the same time brings to an end the receptor impulses (s_u) arising from the current passing through the receptor organs buried in the tissue.

At first thought it might be supposed that since only consistent reinforcement will set up stable habits, and since the reaction (R) when in a given situation yielding S to the receptors will be followed consistently by a reinforcing state of affairs only when there is a causal connection between the antecedent events and those which follow, the "law of effect" would be established on a firm *a priori* foundation. In point of fact, however, this rule breaks down in the Pavlovian conditioned-reflex experiment where the salivary reaction of the dog can by no stretch of the imagination be regarded as the cause of the receipt of food which reduces the hunger and is commonly considered the reinforcing agent in this experiment. This paradox is explained by observing that some *common* cause, the food, produces first the salivation and, later, the reduction in the need (hunger). Accordingly the reinforcing state of affairs is temporally related to the reaction involved in the reinforcement in strict accordance with the law-of-reinforcement formulation, but not as the effect of the reaction being reinforced. A second presumptive exception to Thorndike's formulation of the nature of the reinforcement process is the conditioned kneejerk (10). The termination of the receptor discharge from the slightly injurious blow on the patellar tendon occurs because of the brief duration of the impact of the hammer, rather than because of the occurrence of the kneejerk.

Despite these minor exceptions, Thorndike's inductive generalization, as represented by the expression, "law of effect," is based upon a very penetrating bit of scientific insight into the dynamics of adaptive situations in general. Nevertheless the exception is probably genuine and it has seemed best to employ in the present work the slightly more appropriate though less colorful expression, *law of reinforcement*.

What Is the Critical Factor in Primary Reinforcement?

In Figures 13, 14, and 16 it will have been observed that reductions in (1) the need and (2) the receptor response to the need *both* follow as consequences of the act involved in the reinforcement process, directly in Figures 13 and 14 and indirectly in Figure 16. These considerations raise the question as to which of the two is to be regarded as the critical reinforcing agent; this can be determined only when some radical experiment is performed in which one of the two is eliminated and the other remains active. The writer is aware of no critical evidence of this kind. Until such becomes available the issue must remain uncertain. Meanwhile, in the interest of definiteness, the alternative of reduction in drive-receptor response is chosen for use in the present work as the more probable of the two. Should critical evidence later prove this choice to be in error, a correction can be made. In the present stage of our ignorance regarding behavior dynamics,³ an error in either direction would not seem to have such far-reaching systematic implications as to render correction unduly difficult.

The Effectiveness of Reinforcement and the Intensity of the Need Involved in the Reinforcement

A recent study by Finan (4) tends to support the view that reduction of need is a critical factor in the primary reinforcement process. This investigator

trained groups of albino rats to secure pellets of food by depressing a small bar in a Skinner-Ellson apparatus (see pp. 87, 268). Each group of animals received the same number of reinforcements but in a different condition of food privation. Two days later, after the food need had been equalized, all groups were extinguished. The median number of non-reinforced reactions required to produce a constant degree of experimental extinction were as follows:

Hours of food privation during reinforcement	1	12	24	48
Median number reactions to produce extinction	25	57.5	40	41

While the above extinction values indicate that the relationship is not a simple increasing function of the number of hours' food privation at the time of reinforcement, they do show that for some hours after satiation there is a progressive increase in the effectiveness of reinforcement; thus the two phenomena are shown definitely to be connected.

The Onset, Versus the Termination, of Need-Receptor Impulse as the Critical Primary Reinforcing Factor

The view has been put forward in the preceding pages that the termination of need-receptor impulse is the critical factor in the primary reinforcement process. Many students in this field, however, have held the view that reinforcement is critically associated with the *onset* of the need or drive, as represented by the physiological shock in Demonstration Experiment C.

The evidence from innumerable selective learning experiments, as typified by Demonstration Experiments A and B, leaves little doubt as to the soundness of the need-reduction generalization. This does not necessarily mean that the need-onset hypothesis is false; there may be more than one mechanism of reinforcement. While such seeming improvidence in biological economy appears somewhat opposed to the principle of parsimony, it is not without parallel in other fields; most organisms possess more than one means of excretion, and some organisms possess more than one independent means of reproduction. Such general considerations merely pose the question and warn us of multiple possibilities; they cannot be decisive.

Turning to experimental evidence now available we find that selective learning of the type shown in Demonstration Experiments A and B usually yields results consistent only with the termination hypothesis. On the other hand, the results from conditioned-reflex experiments, typified by Demonstration Experiment C, are consistent with either hypothesis. This ambiguity probably arises from the brief duration of the shock usual in such experiments; the onset and termination of the need occur so close together that it is difficult clearly to distinguish the influence of each. For example, it is quite possible that the critical reinforcing factor in the conditioned kneejerk experiment (10) often cited in this connection (5, p. 85) may be the termination of the receptor discharge resulting from the usually rather severe blow on the patellar tendon, which is the unconditioned stimulus used to evoke this reaction. The matter is complicated still further by the intrusion of the generalized results of previous learning, especially where highly sophisticated human subjects are employed in the investigations. Thus the only critical evidence now available seems to favor the reduction or termination hypothesis; decision must probably come from carefully designed and executed

experiments perhaps involving surgical interference with portions of the nervous system.

In the interim we shall proceed on the positive assumption that the termination of the need (or of its closely correlated receptor response) is a primary reinforcing factor; this hardly seems open to doubt. Even if the onset of the need, or of the correlated receptor response, proves to have genuine reinforcing capacity, the dynamics of behavior are such that it would not have much adaptive value.

REFERENCES

1. BECHTEREV, V. M. *General principles of human reflexology* (trans. by E. and W. Murphy from the Russian of the 1928 ed.). New York: International Publishers, 1932.
2. CANNON, W. B. *The wisdom of the body*. New York: W. W. Norton and Co., 1932.
3. DASHIELL, J. F. *Fundamentals of general psychology*. New York: Houghton Mifflin Co., 1937.
4. FINAN, J. L. Quantitative studies in motivation. I. Strength of conditioning in rats under varying degrees of hunger. *J. Comp. Psychol.*, 1940, 29, 119-134.
5. HILGARD, E. R., and MARQUIS, D. G. *Conditioning and learning*. New York: D. Appleton-Century Co., Inc., 1940.
6. HULL, C. L. Mind, mechanism, and adaptive behavior. *Psychol. Rev.*, 1937, 44, 1-32.
7. MURPHY, G. *A briefer general psychology*. New York: Harper and Brothers, 1935.
8. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
9. THORNDIKE, E. L. *The fundamentals of learning*. New York: Teachers College, Columbia Univ., 1932.
10. WENDT, G. R. An analytical study of the conditioned kneejerk. *Arch. Psychol.*, 1930, 19, No. 123.

CHAPTER VII

The Acquisition of Receptor-Effector Connections— Secondary Reinforcement

The sequences of learned behavior considered in the last chapter were all very short when perfected, only two or three seconds at the most being required for their execution. These examples were chosen not because they were especially typical of mammalian behavior in general, but because they were relatively simple and so lent themselves readily to an introductory exposition of the principles of learning. We must now explicitly recognize the fact, confirmed by universal observation of the everyday behavior of animals including ourselves, that a great deal of behavior takes place in relatively protracted sequences in which primary reinforcement normally occurs only after the final act. Evidence will be presented in a subsequent chapter (p. 139 ff.) showing that reinforcement probably must follow a receptor-effector conjunction ($\dot{s}C_E$) within about twenty seconds if it is to have an appreciable effect. Consequently direct or primary reinforcement, as such, is inadequate to account for a very great deal of learning. Fortunately an ingenious series of experiments performed in Pavlov's laboratory in Petrograd has yielded a principle which explains these more remote reinforcements. This supplementary reinforcement principle is called *secondary reinforcement*. In the present chapter we shall consider the nature, origin, and elementary functioning of this extremely important principle.

DEMONSTRATION OF THE EXISTENCE OF SECONDARY REINFORCEMENT

Because the principle of secondary reinforcement was first isolated from the results of conditioned-reflex experiments, we shall begin our presentation with an illustrative example from Pavlov's laboratory. The experiment was performed by Dr. Frolov; the only account of this experiment available to English readers is that of Pavlov (7, p. 34), who unfortunately omitted many of the

details necessary to an introductory account of this type of experimentation. For the benefit of those unfamiliar with the methodologies of conditioned-reflex laboratories, these accessory details are here supplied from the accounts of other relevant experiments.

Dr. Frolov experimented with a dog, one of whose salivary glands had been diverted surgically so that the saliva discharged through a fistula in the side of the animal's face instead of flowing into its mouth. Suitable apparatus was provided for the precise determination of the number of drops secreted within a given time interval. When hungry this dog would be presented with the ticking of a metronome for a minute or so, and after 30 seconds meat powder would (presumably) be blown into its mouth; the powder would then be eaten by the dog, a considerable quantity of saliva evoked by the incidental gustatory stimulation and chewing activity at the same time flowing from the fistula. After numerous reinforcements of this kind it was found that the metronome acting alone for 30 seconds evoked 13.5 drops of saliva; this is an ordinary or "first-order" conditioned reflex. The above account presents a fairly typical picture of conditioned-reflex learning by the Pavlovian technique.

Next, a black square was presented in the dog's line of vision for the first time; no saliva flowed from the fistula during this stimulation. Following this test the black square was held in front of the dog for 10 seconds, and after an interval of 15 seconds the metronome was sounded for 30 seconds, *no food being given*. The tenth presentation of the black square (alone) lasted 25 seconds; during this period 5.5 drops of saliva were secreted. This is an example of a "higher-order" conditioned reflex.

The conditions of Frolov's experiment show that the visual stimulation resulting from the presentation of the black square had in some way acquired from association with the metronome stimulation the capacity to evoke the salivary secretion independently. Since the presentation and consumption of food were not associated with the acquisition of the second conditioned reaction, it is assumed that during the original conditioning process the metronome had not only acquired the capacity to evoke the flow of saliva but had also acquired the capacity itself to act as a reinforcing agent. The metronome is accordingly said to be a *secondary reinforcing agent*. For analogous reasons the resulting receptor-effector connection (black square \longrightarrow salivation) set up by this means is said to be a *second-order* conditioned reaction.

SOME PROBLEMS CONCERNING SECONDARY REINFORCEMENT

Frolov's experiment demonstrates in an unambiguous manner the genuineness of secondary reinforcement, a first-rate scientific achievement. Unfortunately, even when considered together with the other Russian experiments in this field it leaves unanswered numerous questions concerning the conditions necessary and sufficient for secondary reinforcement to occur.

1. The reaction conditioned to the black square was qualitatively the same as, though weaker than, that previously conditioned to the metronome. Is this typical of secondary reinforcement, i.e., is secondary reinforcement confined to the transfer of the *same* reaction from one stimulus to another, or may *any* receptor-effector conjunction be connected by secondary reinforcement?

2. In Frolov's experiment the metronome purports to have served a double function: (a) that of *evoking* the reaction (salivation) which was secondarily conditioned; and (b) that of *reinforcing* the conjunction of the salivation thus evoked and the receptor discharge produced by the presentation of the black square. Is this apparent duplication of function by the metronome genuine and, if so, is it a characteristic or necessary part of the secondary reinforcement process?

3. The receptor-effector conjunction involved in the setting up of this second-order conditioned reflex was associated temporally not only with the stimulation of the ticking metronome, but also with the evocation of the reaction already conditioned to the latter. This leads us to ask: Are both of these events, i.e., both the presentation of the metronome stimulus and the evocation of the reaction conditioned to it at the time it acquired the power of being a secondary reinforcing agent, necessary for the secondary reinforcement to occur, or is only one of these events necessary for secondary reinforcement, and if only one, which one?

These and a number of other questions of a somewhat similar nature arising from the Russian experiments in this field must be examined. Before doing this, however, one or two general remarks may be made concerning the situation as a whole. To a certain extent these questions arise because of the distinctly artificial nature of the conditioned-reflex experimental procedure. While in no way detracting from the scientific significance of these investigations, their artificiality probably does in some cases interfere with the recognition of their bearing on the adaptive dynamics of ordinary life situations. Accordingly, while considering the problems in question, an attempt will be made gradually to place the subject of secondary reinforcement in a more natural functional perspective.

WHAT REACTIONS MAY BE SECONDARILY REINFORCED?

We shall begin with the first of the above questions—whether the reaction involved in secondary reinforcement must necessarily be the same as that already conditioned to the secondary reinforcing stimulus, or whether any reaction whatever may be so connected. In order to find an answer to this question it will be necessary to consider some experiments which differ considerably from the type employed by Pavlov. The first of these has been reported by Skinner (9, p. 82).

This investigator fed a hungry rat tiny pellets of specially prepared food by activating a food magazine which dropped one pellet into a food cup at each activation. On each occasion the action of the magazine produced a clearly audible sound vibration. In the course of such training rats soon learn to interrupt whatever they are doing when the magazine vibration occurs, go directly to the cup, and eat the pellet. After 60 pellets had been given in this way, the food was removed from the magazine and the rat left to itself.

A horizontal brass bar projected from the wall of the apparatus several centimeters above the food cup. In its explorations around the food cup the still hungry rat was almost certain sooner or later to stand up on its hind legs and rest its front paws on this bar, which was so delicately pivoted that even a light downward pressure would depress it. Moreover, the bar was so connected with the food magazine that this downward movement would activate the food-release mechanism with its characteristic sound vibration. Because of the preceding training this vibration would at once cause the animal to search in the cup for a pellet; however, no pellet would be found, because in this phase of the experiment the food magazine was empty. This being the case it might be supposed that the act of pressing the bar would not be reinforced.

Skinner ran four rats through this experiment and is of the opinion that the click really did reinforce the bar-pressing act as contrasted with innumerable other acts evoked by the situation. A record made by one of these animals is reproduced as Figure 18. Each small unit in the rise of this curve represents one operation of the lever by the rat. It may be seen that the reactions, as shown by the slope of the curve, occurred with about maximum frequency at the outset of the process and then gradually ceased as the curve

became horizontal. Since no food was given, this learning is presumably the result of secondary reinforcement.

Unfortunately, Skinner publishes no account of an appropriate control experiment to show how frequently a comparable animal would depress the bar if the sound of the action of the magazine had also been eliminated. Nevertheless, much corroborative evidence from other investigations supports Skinner's view that genuine learning took place.

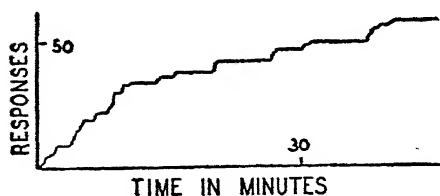


FIG. 18. Record of secondary-reinforcement learning in which the act learned (pressure on a bar) was distinct from that primarily conditioned (putting head down to food cup, salivating, etc.). (After Skinner, 9, p. 83.)

animals were extinguished¹ by so adjusting the apparatus that pressure on the bar was no longer followed by the delivery of the food pellet. With one group, however, the depression of the bar was followed at once by the customary click of the food-release mechanism, but with the other group it was not. Bugelski found that the click-extinction group, as a whole, executed a little over 30 per cent more pressures on the bar before reaching extinction than did the non-click group. This indicates in a convincing manner the power of a stimulus (the magazine click) closely associated with the receipt of food to contribute to the maintenance of a receptor-effector connection at a superthreshold level.

The Skinner and Bugelski studies, taken jointly, enable us to answer the first two of the questions raised by the Russian secondary-reinforcement experiments. On the analogy of the Pavlovian type of conditioned-reflex experiment, the vibrations of the food-release apparatus in Skinner's experiment may be assumed to have become conditioned to salivary secretion and other phases of the

¹ The subject of extinction will be taken up in detail in a later chapter (p. 258 ff.). For the present it may merely be said that when a learned reaction is repeatedly evoked and the evocation is not followed by reinforcement, the stimulus gradually loses its capacity to evoke the reaction. This loss is known as *experimental extinction*.

eating process, as, clearly, was the tendency to put the head down to the cup and search for the food, by *primary* reinforcement.¹ On the other hand, the act *secondarily* reinforced in the Skinner experiment was that of depressing a bar. Two acts could hardly be more diverse than sniffing in a food cup and depressing a bar with the paws. We conclude, then, that the identity of the act in the primary and the secondary reinforcement of the Russian experiments was an accidental condition and that such an identity is not a necessary characteristic of secondary reinforcement in general. Thus the first of the above questions is answered in the negative.

The two experiments just examined also enable us to answer the second question posed by the Frolov experiment. Since the act secondarily conditioned was clearly different from that conditioned to the secondary reinforcing stimulus, it follows that the double rôle of evoking the reaction to be conditioned and at the same time serving as a reinforcing agent is not necessarily characteristic of secondary reinforcing agents in general.

MUST THE SECONDARY REINFORCING STIMULUS EVOKE ITS
CONDITIONED REACTION IN ORDER TO ACT AS
A REINFORCING AGENT?

The finding of an answer to the third of our questions involves a determination of the differential causal efficacy in secondary reinforcement of two events which usually occur together, namely, the so-called secondary-reinforcing stimulus and its conditioned reaction. The problem could be solved readily enough if we could devise some way of presenting in a situation known to be capable of reinforcement each of the factors separately, and observing whether or not learning does in fact occur in each case. The difficulty lies primarily in accomplishing the complete and certain elimination of the one factor while retaining the complete integrity of the other. While no investigations have been found which were deliberately directed to a solution of this problem, there are two or three which throw indirect light upon it. One of these, reported by Cowles (2), purports to have eliminated from the secondary reinforcement situation at least the gross overt reaction conditioned to the secondary reinforcing stimulus.

Cowles readily trained two chimpanzees to insert 1¼-inch colored disks into a slot machine which delivered a raisin for each

¹ However, see terminal note entitled, "The Status of Food Reward as a Reinforcing Agent."

disk inserted. After this training the animals would retain, hoard, and even expend considerable amounts of energy to secure the disks, evidently as subgoals. Later each animal was presented with the task of learning which of a row of five small, lidded boxes contained concealed within it one of these tokens. Each learning session consisted of 20 trials, all of which had to be completed before the animal could exchange its tokens for raisins at the slot machine which was located in a room about 35 feet distant. Each series of 20 trials was devoted to a separate box, so that old habits of choosing other boxes on previous training series had to be broken, and new ones substituted, at every succeeding session. Finally on alternate sessions, in order to secure a measure of the relative strength of primary and secondary reinforcement, instead of a token real food in the form of a raisin was put in the box. It was found that the average score of the two apes on the second half of each 20 trials on a given box (where chance success alone would yield 20 per cent correct choices) was 74 per cent for food tokens and 93 per cent for the food. This shows an amount of learning due to secondary reinforcement which is fairly comparable to that mediated by the primary food reward itself.

In this experiment the overt act normally evoked by the secondary reinforcing agent, the food token, was that of inserting the token in the slot machine, whereas the act involved in the process of secondary reinforcement was that of lifting the lid of a particular box in a row of five. Under the conditions of this multiple-choice learning the slot machine was in a different room, and the animals literally could not carry out the act of inserting the token for some time after obtaining it. Moreover, Cowles reported no tendency on the part of the apes to execute movements such as to insert the disks into an imaginary vending machine. In so far the Cowles experiment seems to yield a negative answer to the third query suggested by Frolov's experiment; i.e., it suggests that the occurrence of the first-order conditioned stimulus is necessary for the setting up of a second-order conditioned reaction, and that the occurrence of the primary conditioned reaction is not necessary.

THE EFFECT OF EXTINCTION OF THE PRIMARY RECEPTOR-EFFECTOR CONNECTION ON SECONDARY REINFORCEMENT

Despite the results from Cowles' experiment, it would be rash to conclude at once that the evocation of some fractional compo-

ment of the reaction originally conditioned to the secondary reinforcing stimulus was not present at each secondary reinforcement. The principles of reinforcement learning lead *a priori* to the expectation that salivation, and probably many other hidden internal processes such as the galvanic skin reaction, must have been conditioned both to the stimulus energies arising from the vending machine and to those from the food tokens. Moreover, we saw above that in Frolov's experiment salivary secretion accompanied every secondary reinforcement of the black square. The fact that such processes were not observed in Cowles' experiment argues little against the formidable probability that they did in fact occur. Had not special apparatus and procedures been employed, the salivary secretion would not have been observed in Frolov's experiment either. On the positive side, Cowles (2) reports that the apes employed in his experiments showed a marked tendency to put the food tokens in their mouths. Wolfe (12, p. 16) reports that both food and food tokens would elicit anticipatory lip-smacking activity, but a brass non-food token of the same shape would not. It is clear from the above considerations that further evidence will be required to determine whether the presence of the reaction component of the usual secondary reinforcing situation is a necessary condition for the occurrence of secondary reinforcement.

We saw above in connection with the Bugelski experiment (p. 88, footnote) that receptors which frequently evoke a conditioned reaction without accompanying reinforcement presently lose the power of evoking this reaction. Experimental extinction accordingly offers a means of separating a secondary reinforcing stimulus from the reaction to which it was conditioned while it was acquiring its secondary reinforcing powers. This circumstance makes possible the presentation of the former without the latter in close temporal proximity to a reinforceable receptor-effector conjunction. Such a combination of circumstances occurred in a quantitative experiment reported by Grindley (3).

In one of his experiments Grindley placed young chickens at the beginning of a four-foot runway. At the other end were placed grains of boiled rice. Half of the chickens were permitted to find their way down the runway and eat the rice. The other half were likewise permitted to go down the runway, but found a plate of glass placed a few inches above the rice which prevented them from eating the grains. An index based on the time required by each of the two groups of chickens to traverse the runway on each of

the 12 successive trials is shown in Figure 19. Our interest is confined mainly to the scores of the group of chickens which saw but could not eat the rice. This curve shows that for the first four or five trials the non-eating chickens gained in speed nearly as fast as did the rewarded ones. During the subsequent trials, however, the non-rewarded chickens gradually lost their rate of locomotion until at the eleventh and twelfth trials they did scarcely

better than the chance performance of the very first trial.

These results of Grindley's experiment are duplicated by Skinner's record (Figure 18), which is so constructed that the flattening out of his curve to a horizontal represents experimental extinction after what appears to be a rather abrupt learning. Pavlov reports the same phenomenon in the conditioned-reflex learning situation; extinction was encountered by him especially when he attempted to set up third and fourth order conditioned reflexes.

The experimental results just considered, as well as those from numerous concordant experiments of both

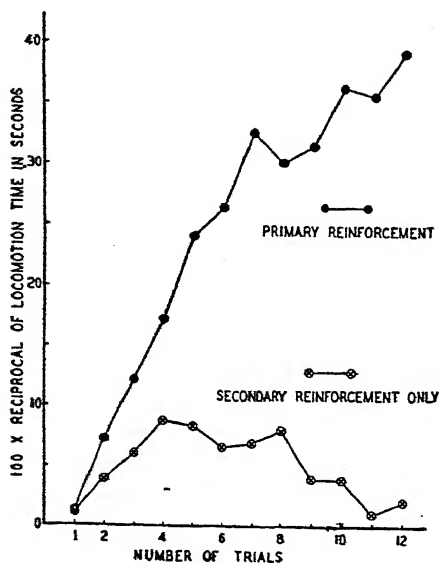


FIG. 19. Graphs showing in parallel the course of the learning of young chickens to traverse a straight four-foot runway by primary and by secondary reinforcement which was unaccompanied by the original supporting primary reinforcement. (After Grindley, *S*, p. 179.)

conditioned-reflex and selective learning, all point to the same conclusion, namely, that a secondary reinforcing agent (in Grindley's experiment the visual stimulus presented by the rice grains) loses its power of secondary reinforcement when it loses its power of evoking the reaction conditioned to it at the time it acquired its power of secondary reinforcement; moreover, it would appear to possess the power of reinforcement only to the degree that it possesses the power of evoking this reaction. It follows that in cases in which the secondary reinforcing agent is still strong, secondary reinforcement

makes some progress. Very soon, however, it weakens through experimental extinction, reinforcement is thereby withdrawn, and the secondary reinforcement declines along with the strength of its parent receptor-effector connection.

Granting that the occurrence of at least some component of the reaction conditioned to the stimulus in the usual secondary reinforcing situation is necessary for secondary reinforcement to occur, our original question is still far from answered. Even if the reaction is necessary, this does not mean that it is sufficient. This confronts us with the question of whether the stimulus also is necessary. The empirical solution of the latter problem would require that the reaction conditioned to the stimulus of a secondary reinforcing situation somehow be presented without the evoking stimulus, in close temporal proximity to a reinforcing receptor-effector conjunction, and that a determination be made as to whether or not learning takes place. No experiments in which this was attempted have been found. Judgment in this intricate but theoretically important matter must accordingly be held in abeyance until more adequate evidence becomes available.

THE POSSIBILITY OF SECONDARY REINFORCEMENTS ABOVE THE SECOND AND THIRD ORDERS

A fourth question raised by the Russian experiments on secondary reinforcement concerns the possibility of setting up conditioned reactions above the second order. In discussing this question Pavlov remarks (7, pp. 34-35):

It was found impossible in the case of alimentary reflexes to press the secondary stimulus into our service to help us in the establishment of a new conditioned stimulus of the third order. Conditioned reflexes of the third order can however be obtained with the help of the second order of conditioned reflexes in defence reactions such as that against stimulation of the skin by a strong electric current. But even in this case we cannot proceed further than a conditioned reflex of the third order. . . . In these conditioned reflexes, passing from the first to the third order, the latent period progressively increases. In the same order we pass from the strongest to the weakest conditioned defence reflex.

The above passage from Pavlov has been quoted at length because there is reason to believe that while the facts there reported are well authenticated, their adaptive implications have occasion-

ally been misunderstood, possibly even by Pavlov himself. This presumptive error in interpretation seems to have come about through the distinctly narrow and artificial nature of the conditioned-reflex experimental procedure by means of which it was investigated, much as is believed to have been the case in the determination of what constitutes a primary reinforcing state of affairs (p. 78). In order to prove that secondary reinforcement is a genuine phenomenon it is necessary to remove all possibility of primary reinforcement from the situation. This, of course, incidentally causes extinction of the primary receptor-effector connection, which, as we saw in Grindley's experiment, soon leads to the loss of the power of reinforcement by the secondary reinforcing agent and thus tends to bring the potential chain of transfers of the power of reinforcement to an early termination. As indications of this we note Pavlov's statement that each successive reaction in the chain had a progressively longer latency, itself an indication of receptor-effector weakness which is also separately noted by him. Viewed in the light of these considerations the limitation in the number of higher-order conditionings was presumably a mere artifact of the technical procedure employed in the investigation of the problem.

We now have evidence which indicates that the gradient of reinforcement does not extend backward from the reinforcing state of affairs in detectable amounts beyond about 20 seconds (8). This means that in protracted behavior sequences, even with primary reinforcement fully intact, the direct effects of the latter are automatically excluded for all receptor-effector conjunctions beyond a half minute or so from the point of such reinforcement. Since in the higher organisms behavior sequences, e.g., the pursuit of prey, often continue far beyond such a limit, it follows that the strength of the earlier segments of such sequences must frequently be maintained by very long chains of secondary reinforcing situations. In normal human organisms it would appear from such considerations that higher-order conditioning yields receptor-effector connections which are comparable in vigor with those produced by primary reinforcement, and that there is practically no limit to the degree to which higher-order conditioning may be carried under suitable conditions. As for the latter, it may be said that with the exception of the nature of the reinforcing state of affairs involved the conditions necessary for secondary reinforcement are the same as those for primary reinforcement. This is to say that *a receptor*

impulse will acquire the power of acting as a reinforcing agent if it occurs consistently and repeatedly within 20 seconds or so of a functionally potent reinforcing state of affairs, regardless of whether the latter is primary or secondary.

THE RÔLE OF SECONDARY REINFORCEMENT IN COMPOUND SELECTIVE LEARNING

As a means of showing something of the systematic and functional significance of secondary reinforcement we shall now consider a few of its more elementary implications. It has already been suggested (p. 84) that secondary reinforcement plays its major rôle in protracted behavior sequences, particularly in those portions of them which precede the point of primary reinforcement by more than 20 seconds or so. A typical sequence of this kind is found in compound selective learning. This may be thought of as a series of trial-and-error learning situations in which each link in the series consists of a simple selective learning situation possessing the general characteristics of Demonstration Experiment A. It may further be assumed that at the beginning of learning 20 seconds or more are consumed by the organism before the correct reaction is performed at each of five choice points; that the correct reaction in one situation always leads at once to the next situation in an invariable order; that the new situation instantly activates the receptors of the organism in a distinctive way; and that the entire sequence finally culminates in the complete satiation of the need which motivated the organism throughout the total activity. Following common-sense usage, we shall call this final primary reinforcing state of affairs the *goal* and represent it by the letter *G*. If there are five segments in such a behavior sequence, we may represent the correct reaction of the first trial-and-error situation by R_1 , that of the second by R_2 , and so on to R_5 . In a similar manner the gross stimuli presented to the organism by the respective situations may be represented by the parallel notation, S_1 , S_2 , S_3 , S_4 , and S_5 .

It follows from the conditions of compound selective learning assumed above that the organism by sheer trial-and-error will work blindly down through the series until $S_5 \longrightarrow s_5$ evokes R_5 , which by physical causation produces G , the primary reinforcing state of affairs. This, by the principle of primary reinforcement, begins to set up the connection $S_5 \longrightarrow s_5 \dashrightarrow R_5$ and at the same

time, by the principle of secondary reinforcement, to endow S_5 with the powers of (indirect) reinforcement. Now, R_4 is too remote in time from G to be selected by primary reinforcement. However, in the course of subsequent executions of the sequence by the organism the *secondary* reinforcing power recently acquired by S_5 will mediate the connection $S_4 \longrightarrow s_4 \dashrightarrow R_4$, at the same time endowing S_4 with secondary reinforcing power. In the same way secondary reinforcement will move progressively backward from S_4 to S_3 , from S_3 to S_2 , and finally from S_2 to S_1 , ultimately resulting in the tightly knit, errorless behavior sequence shown in Figure 20.

It is clear from the preceding that compound selective learning must necessarily progress in a backward manner from the point of primary reinforcement. This means that in the situation under consideration errors would be eliminated most quickly at S_5 , next

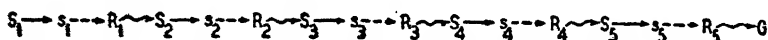


FIG. 20. Diagrammatic representation of the final phase of a case of compound selective learning in which simple trial-and-error occurs five times. If at the beginning of learning the occurrence of each correct R be assumed to consume at least 20 seconds then the four segments at the left of the figure would be dependent for their selection purely upon secondary reinforcement. In that event S_2 would be the secondary reinforcing agent effecting the selection of R_1 , and S_3 would be the secondary reinforcing agent effecting the selection of R_2 . As in Figures 13, 14, and 16, the sign $\sim\sim\sim$ represents a non-physiological causal connection.

most quickly at S_4 , and most slowly of all at S_1 , the rate-of-learning score at the several points presenting a gradient whose highest point would be at S_5 . That such a gradient exists in compound selective learning situations is well known, though it may be overridden by various known factors. Because of the relation of this gradient to the point of primary reinforcement or goal, the backward order of the elimination of errors has been called the *goal gradient* (5). We shall recur to this subject in another connection (p. 142).

There should also be noted the implication in the above analysis that certain circumstances in the learning situation may effectively prevent learning from occurring, particularly in the segments anterior to $S_5 \longrightarrow s_5 \dashrightarrow R_5$. One of the most important of these is a delay in the occurrence of S_5 , say, following reaction R_4 . The principles developed earlier in the chapter imply that in an ideal situation in which S_1 , S_2 , S_3 , S_4 , and S_5 are all *totally* different, a delay of 20 seconds or more will prevent learning at any point in

the series anterior to S_3 . On the other hand, if S_1 , S_2 , S_3 , and S_4 all contain an important component found in S_5 (a situation easily set up experimentally) then each will become a reinforcing agent regardless of the temporal gaps in the series.

SUMMARY

Primary reinforcement, because the range of its gradient is limited to 20 or 30 seconds, is incapable of explaining the learning which manifestly occurs when the receptor-effector processes involved are temporally very remote from the relevant need reduction. These latter reinforcements are explained by the discovery that the power of reinforcement may be transmitted to any stimulus situation by the consistent and repeated association of such stimulus situation with the primary reinforcement which is characteristic of need reduction. Moreover, after the reinforcement power has been transmitted to one hitherto neutral stimulus, it may be transferred from this to another neutral stimulus, and so on in a chain or series whose length is limited only by the conditions which bring about the consistent and repeated associations in question. The inability of Pavlov and his pupils to obtain conditioned reactions above the third order appears to have been due to the highly artificial nature of their experimental procedures which did not provide the necessary conditions for long and stable secondary-reinforcement chains.

Our detailed findings concerning secondary reinforcement may be listed as follows:

1. Perhaps the most striking characteristic of secondary reinforcement is that it is itself a kind of by-product of the setting up of a receptor-effector connection, in the first instance through primary reinforcement. Primary reinforcement, on the other hand, appears to be a native, unlearned capacity in some way associated with need reduction.
2. Secondary reinforcement may be acquired by a stimulus from association with some previously established secondary reinforcement, as well as with a primary reinforcement. It would appear that transfer of this power of reinforcement from one stimulus situation to another may go on indefinitely, given the conditions of stable and consistent association.
3. A receptor-effector conjunction involving any effector may be reinforced by any secondary reinforcing situation.
4. Secondary reinforcement differs from primary reinforcement in that the former seems to be associated, at least in a molar sense, with stimulation, whereas the latter seems to be associated with the cessation of stimulation, i.e., of the S_D .
5. Stimuli which acquire secondary reinforcing power seem always to

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acquire at the same time a conditioned tendency to evoke an associated reaction. The available evidence indicates that as such stimuli lose through extinction the power of evoking this reaction, they lose in about the same proportion their power of secondary reinforcement. It is probable also that the reverse is true; that a stimulus gradually acquires its powers of secondary reinforcement as it acquires the power of evoking the reaction conditioned to it.

6. It follows from (5) that a stimulus alone is ineffective as a secondary reinforcing agent. We do not know whether the reaction evoked by this stimulus, or possibly some critical fraction of that reaction if presented without the stimulus, would serve as a reinforcing agent. Conceivably, of course, the combination of *both* the stimulus and its conditioned reaction, or some fractional component thereof, is necessary to effect secondary reinforcement.

7. It is apparent from the preceding that a reaction conditioned to a stimulus which has as a by-product acquired secondary reinforcing power, at a point several secondary-reinforcement links removed from the point of primary reinforcement, may suffer experimental extinction in two ways: (a) through the evocation of the reaction not being followed by the parent secondary-reinforcing stimulus, and (b) through the evocation of the reaction being followed by the parent stimulus after the latter has lost its secondary reinforcing power through, say, the extinction of its own conditioned reaction.

8. With the main facts of secondary reinforcement before us we may now reformulate the law of reinforcement in such a way as to include the wider learning potentialities inherent in secondary reinforcement:

Whenever an effector activity occurs in temporal contiguity with the afferent impulse, or the perseverative trace of such an impulse, resulting from the impact of a stimulus energy upon a receptor, and this conjunction is closely associated in time with the diminution in the receptor discharge characteristic of a need or with a stimulus situation which has been closely and consistently associated with such a need diminution, there will result an increment to the tendency for that stimulus to evoke that reaction.

NOTES

The Status of Food Reward as a Reinforcing Agent

We have seen in the preceding pages that primary reinforcement originates in the reduction of a primary need. Now, the primary need in the case of food privation presumably consists in the requirement of the cells of the body for nutriment, just as the primary need in the case of the shocked animals in Demonstration Experiments A, B, and C was the cessation of the action of the electric current on the cells of the animals' feet. It is evident from a knowledge of nutritional physiology that there is an appreciable delay between the beginning of the mastication of food and the ultimate reduction in the nutrient need of the body.

cells while mastication, deglutition, digestion, and absorption are taking place. This makes it distinctly improbable that the presentation of food, its mastication, and the gustatory stimulus incidental to mastication are primary reinforcing states of affairs. Yet innumerable experiments have shown that the presentation and mastication of food in fact constitute a powerful reinforcing combination. These considerations strongly suggest that *the eating of food as such brings about learning through secondary reinforcement rather than through primary reinforcement.*

Incidentally, this hypothesis explains the paradox that whereas other cases of clear primary reinforcement appear to be associated with reduction in drive stimulation ($S_D \rightarrow s_D$), food reinforcement appears at the instant of reinforcement to be associated rather with stimulation or the onset of stimulation which, as we have seen above, is characteristic of secondary reinforcement. This may account for Pavlov's view that reinforcement is primarily a phenomenon of stimulation.

A Possible Technique for Determining the Status of Food Reward as a Reinforcing Agent

Because of the wide use of food reward as a reinforcing agent in behavior experiments its status in this respect is in especially urgent need of clarification. There is reason to believe that a sham feeding experiment might contribute materially to this end. The esophagus of a dog could be severed and both the upper and lower sections converted into fistulas opening through the skin of the dog's neck. With such an arrangement it would be possible to feed the dog and either have the masticated food fall into a conveniently placed receptacle or have it pass through a tube connecting one fistula with the other and thus enter the stomach and ultimately reduce the need of the body cells for nutriment. Since the various receptor discharges associated with the eating of food, its swallowing, digestion, and absorption, have throughout the entire life of each organism been associated in a uniform and practically invariable sequence with ultimate need reduction, it is to be expected that the stimuli associated with mastication would have acquired a profound degree of secondary reinforcing power. For this reason the power of secondary reinforcement ought not to be lost by such stimuli through a moderate amount of experimental extinction. However, if the present hypothesis is sound, a dog sham fed on one kind of food and really fed on an equally reinforcing kind of food should, after a time, show a distinct preference for the food which mediates nutrition and so primary reinforcement; after much training it might even refuse to eat the sham food since, not being reinforced, this activity should suffer experimental extinction. Careful controls would, of course, need to be carried out on such matters as the food preferences possessed by the dog just before the beginning of the experiment. Numerous variants of the above procedure will at once suggest themselves as alternatives in the solution of this extremely important problem.

Are Primary and Secondary Reinforcement at Bottom Two Things or One?

In the present chapter it has been seen that despite well-marked differences there are a number of striking similarities between primary and secondary reinforcement. Perhaps the most notable of the similarities is the fact of reinforcement itself. So far as our present knowledge goes, the habit structures mediated by the two types of reinforcement agents are qualitatively identical. This consideration alone constitutes a very considerable presumption in favor of the

view that both forms are at bottom, i.e., physiologically, the same. It is difficult to believe that the processes of organic evolution would generate two entirely distinct physiological mechanisms which would yield qualitatively exactly the same product, even though real duplications of other physiological functions are known to have evolved.

While the ultimate proof of the essential identity of the two processes, when and if it comes, must be looked for on the physiological rather than on the behavioral level, it is evident that the present development of neurophysiology is quite remote from such an achievement. Meanwhile the urgency of the problem from the standpoint of systematic behavior theory is such as to make an attempt at a workable first approximation to such a proof on a molar level extremely desirable. This would necessarily take the form of a revised inductive generalization which would be sufficiently comprehensive to include both phenomena as special cases of the same rule or law. While no detailed and fully substantiated hypothesis of this nature will be attempted here, a few suggestions are offered which may possibly contribute to the attainment of this end by others.

In the development of this plan the first important fact to be considered is that *the initial secondary reinforcing stimulus acquires its reinforcing power through a process of reinforcement.* Moreover, the process of successive transmission of this power from one stimulus situation to another, backward through a compound selective learning sequence, also appears always to occur in a reinforcement situation in which the secondary reinforcing stimulus acquires a reaction tendency. These considerations suggest rather strongly that the first secondary reinforcing stimulus acquires its power of reinforcement by virtue of having conditioned to it some fractional component of the need reduction process of the goal situation (G , Figure 20) *whose occurrence, wherever it takes place, has a specific power of reinforcement in a degree proportionate to the intensity of that occurrence.*

Let us represent this fractional component of the goal reaction by the symbol g . Referring back to the compound selective learning situation represented in Figure 20, it is evident on the above hypothesis that while $S_5 \rightarrow s_5$ is acquiring its connection to R_5 , the perseverative trace of s_5 is also acquiring a parallel connection to g . It follows that presently, when the connection $S_5 \rightarrow s_5 \rightarrow g$ acquires a superthreshold strength, g , by the principle of stimulus generalization (see p. 183 ff.), will come forward in the series and occur in close conjunction with S_5 and will therefore serve to strengthen $S_4 \rightarrow s_4 \rightarrow R_4$ as well as to condition itself to $S_4 \rightarrow s_4$, thus: $S_4 \rightarrow s_4 \rightarrow g$. In the course of successive trials or repetitions, when this connection ($s_4 \rightarrow g$) becomes of superthreshold strength, the same thing would occur with S_3 , then with S_2 , and finally with S_1 . In this way g , as both a conditionable process and a reinforcing agent, would be passed back through the sequence. There would therefore develop an understandable *modus operandi* for compound selective learning.

Since it takes time for g to become conditioned to a new trace to a degree such that it can act with much strength as a reinforcing agent, there is bound to be considerable delay between its full action at the goal and that at the beginning of the behavior sequence leading to the need reduction. This would, of course, produce the goal gradient, now known to be characteristic of such learned sequences (10, 11).

We now turn to the matter of experimental extinction. Suppose, in case selective learning has occurred at all the five choice points, that the organism executes the complete action sequence, but that the primary reinforcing state of

affairs, G , does not follow the performance of R_5 . It must be supposed that the conditioned g as evoked by $S_5 \rightarrow s_5$ will be weaker than the unconditioned g evoked in connection with the need reduction at G . Therefore it is to be expected that while $S_5 \rightarrow s_5 \rightarrow g$ will somewhat retard the process of extinction of $S_5 \rightarrow s_5 \rightarrow R_5$ (as in Bugelski's experiment, 1), it will nevertheless not suffice to prevent ultimate extinction. In a similar manner the weakening of $S_5 \rightarrow s_5 \rightarrow R_5$ will rapidly bring about the weakening of $S_4 \rightarrow s_4 \rightarrow g$; this will result in a weakening of $S_4 \rightarrow s_4 \rightarrow R_4$, and so on backward throughout the sequence to its very beginning, gradual collapse of the entire behavior sequence occurring as trial follows trial during the extinction process. Presumably generalized extinction effects would contribute to the speed of inhibition throughout such a behavior series, especially where the trials are given in immediate succession (4, pp. 497-499).

It is to be noted in this connection, however, that the present hypothesis does not imply that secondary reinforcement will necessarily suffer experimental extinction when the support of the primary need reduction is withdrawn. If the primary reinforcement has been sufficiently profound for the connection $S_5 \rightarrow s_5 \rightarrow g$ to be very strong, the conditioned g may be intense enough to withstand the inhibition generated by an indefinitely large number of otherwise unreinforced presentations of S_3 , S_4 , or S_5 . Here, apparently, we have the explanation of what Gordon Allport has called the functional autonomy of higher-order conditioned reactions; the g , if sufficiently well conditioned, may be strong enough in reinforcing power to maintain itself through self-reinforcement—a true functional autonomy. It is probable that something of this kind is operative in certain cases of neurotic symptoms, as has been pointed out by Mowrer (6) in his behavioristic interpretation of Freud's doctrine of anxiety.

REFERENCES

1. BUGELSKI, R. Extinction with and without sub-goal reinforcement. *J. Comp. Psychol.*, 1938, 26, 121-133.
2. COWLES, J. T. Food-tokens as incentives for learning by chimpanzees. *Comp. Psychol. Monog.*, 1937, 14, No. 5.
3. GRINDLEY, G. C. Experiments on the influence of the amount of reward on learning in young chickens. *Brit. J. Psychol.*, 1929, 20, 173-180.
4. HULL, C. L. Goal attraction and directing ideas conceived as habit phenomena. *Psychol. Rev.*, 1931, 38, 487-506.
5. HULL, C. L. The goal gradient hypothesis and maze learning. *Psychol. Rev.*, 1932, 39, 25-43.
6. MOWRER, O. H. A stimulus response analysis of anxiety and its role as a reinforcing agent. *Psychol. Rev.*, 1939, 46, 553-565.
7. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
8. PERIN, C. T. A quantitative investigation of the delay-of-reinforcement gradient. Ph.D. thesis, 1942. On file Yale Univ. Library.
9. SKINNER, B. F. *The behavior of organisms*. New York: D. Appleton-Century Co., Inc., 1938.
10. SPENCE, K. W. The order of eliminating blinds in maze learning by the rat. *J. Comp. Psychol.*, 1932, 14, 9-27.
11. WOLFE, J. B. The effect of delayed reward upon learning in the white rat. *J. Comp. Psychol.*, 1934, 17, 1-21.
12. WOLFE, J. B. Effectiveness of token-rewards for chimpanzees. *Comp. Psychol. Monog.*, 1936, 12, No. 5.

CHAPTER VIII

The Symbolic Construct ${}_sH_R$ as a Function of the Number of Reinforcements

In the course of the preceding discussions of reinforcement the reader may have noticed two implicit assumptions: (1) the receptor-effector connections so set up correspond roughly to what are known to common sense as habits;¹ and (2) the process of habit formation consists of the physiological summation of a series of discrete increments, each increment resulting from a distinct receptor-effector conjunction (${}_sC_R$) closely associated with a reinforcing state of affairs (G). It shall be our task in the present chapter to try to tease out of a series of relatively simple and reasonably well-studied habit-formation situations at least a first approximation to the central law or functional relationship of habit strength as dependent upon the number of these reinforcement increments.

As a preliminary to this undertaking it is important to note that habit strength cannot be determined by direct observation, since it exists as an organization as yet largely unknown, hidden within the complex structure of the nervous system. This means that the strength of a receptor-effector connection can be determined, i.e., can be observed and measured, only indirectly. There are two groups of such observable phenomena associated with habit: (1) the antecedent conditions which lead to habit formation, and (2) the behavior which is the after-effect or consequence of these antecedent conditions persisting within the body of the organism. As our analysis progresses we shall find that habit strength depends upon various antecedent factors in addition to the number of reinforcements. We shall also note that habit strength may manifest itself in several different measurable ways. One of these, the magnitude of the evoked reaction, will next be considered.

¹Strictly speaking, by common usage the referent of the term "habit" is a well-worn mode of *action*, whereas by the present usage the referent is a *persisting state of the organism* (resulting from the reinforcement) which is a necessary, but not a sufficient, condition for the evocation of the action in question.

HABIT STRENGTH AND REACTION MAGNITUDE

The progressive increase in the magnitude of an evoked habitual reaction with successive reinforcements is conveniently illustrated by a study reported by Hovland (1). This investigator associated the Tarchanoff galvanic skin reaction, originally evoked by a mild electric shock on the wrist, with the simple sinusoidal vibrations of

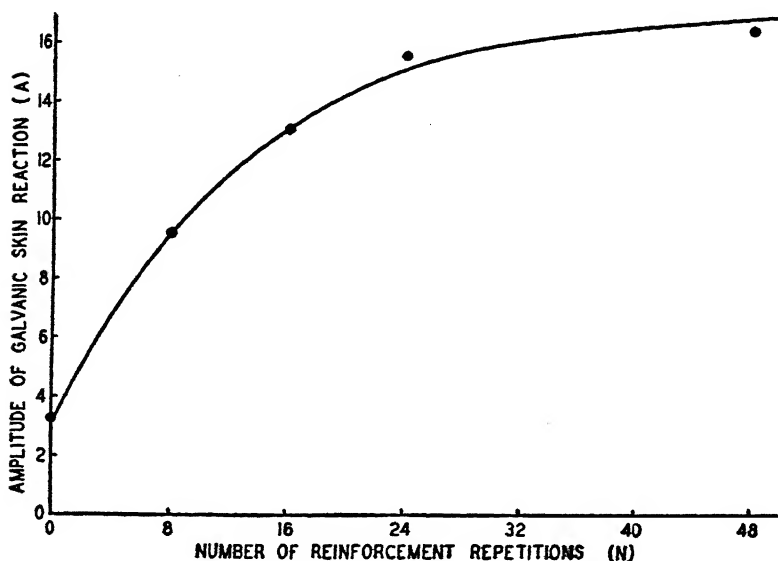


FIG. 21. An empirical learning curve plotted in terms of the amplitude in millimeters of the first galvanic skin reaction evoked by the conditioned stimulus after varying numbers of reinforcements. The circles represent mean readings for different but comparable groups of subjects. The curved line was plotted from values secured by substituting in an equation fitted to the data represented by the circles. (From data published by Hovland, 1, p. 268.)

a beat-frequency oscillator. The galvanic reaction was picked up from the skin of the subject's hand by a pair of polished silver disk electrodes, one bound to the palm and the other to the back. The current thus secured was passed through a sensitive galvanometer, the magnitude of the subject's electrical reaction being shown by the amount of movement across a screen made by a beam of light reflected from a mirror in the instrument. This movement was recorded graphically and was subsequently measured in millimeters. Four matched groups of 32 subjects each were given 8, 16, 24, and 48 reinforcements respectively. At once following this series of

reinforcements the tone was presented alone, and the amplitude (A) of the evoked reaction was measured. The means of the conditioned reactions thus evoked from the several groups of subjects are shown by the circles in Figure 21. The circle at zero is the average amplitude of reactions evoked from all groups of subjects previous to reinforcement. The curved line drawn among the data points represents a series of values calculated from a growth function which has been fitted to the data represented by the circles.

From an inspection of Figure 21 three observations may be made:

1. The value of this reaction at zero reinforcements is not itself zero but has, on the contrary, the very appreciable value of 3.16 millimeters. This is quite characteristic, since it has long been known that the galvanic skin reaction is evocable in considerable amounts by any stimulus of appreciable intensity.
2. The greater the number of reinforcements (and, presumably, the stronger the habit), the greater will be the amplitude of the evoked reaction. Accordingly, the amplitude of the reaction is said to be an *increasing* function of the number of reinforcements.¹
3. Despite a certain amount of deviation of the circles from the fitted curve, possibly due to the limited number of data from which the means were calculated, the relationship appears to approximate rather closely a simple positive growth function.

HABIT STRENGTH AND REACTION LATENCY

A second way in which habit strength may manifest itself in a measurable manner is in the length of time elapsing from the onset of the stimulus to the onset of the associated reaction (st_R). This time interval is called *reaction latency*.

The general relationship of habit strength (as indicated by the number of reinforcements) to reaction latency is illustrated by an investigation reported by Simley (5). In this study college students associated nonsense characters, presented for five seconds each by means of an automatic exposure apparatus, with nonsense syllables presented orally by the experimenter in the middle of each exposure. The subjects were instructed to speak each syllable just as quickly as possible after the corresponding character was presented. A voice key connected with other automatic devices made possible the determination of each reaction latency as the

¹This statement holds for certain reactions, such as salivary secretion and the galvanic skin reaction, but apparently not for all (see p. 329 ff.).

learning progressed. Learning was continued long after the habits involved in any given rote series had passed the reaction threshold. Out of a large number of such stimulus-response combinations, one subject (M.W.) was found to have spoken the associated syllable before being prompted at the second presentation of about 125 of the characters. The mean latencies of these reactions at the second and each of the following fifteen reinforcements¹ are shown by the circles in Figure 22. As in the case of Figure 21, a function has been fitted to these data; this is represented by the curve which passes among the circles.

An inspection of Figure 22 shows:

1. There is no circle representing a latency value at zero reinforcement; this is because no reaction of this complex type can occur previous to any learning. Strictly speaking, this means that the latency of such a reaction is infinite when reinforcement is zero.

2. Reaction latency is inversely related to the number of reinforcements; i.e., the greater the number of reinforcements (and, presumably, the stronger the habit), the shorter the time required for reaction evocation. Thus reaction latency is said to be a *decreasing* function of the number of reinforcements.²

3. Quite as in Figure 21, the data values deviate appreciably from the

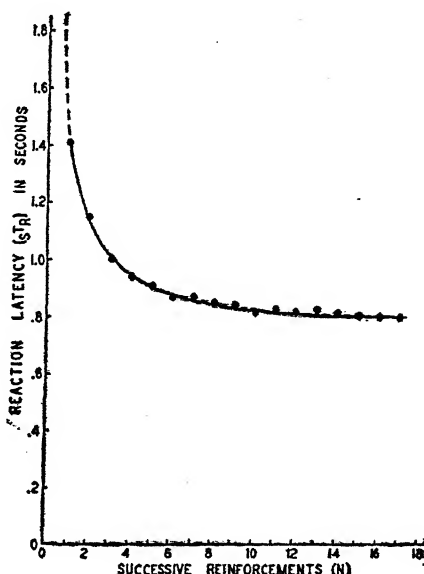


FIG. 22. Empirical learning curve plotted in terms of the reaction latency of speaking nonsense syllables at the presentation of the nonsense character with which they were paired. The circles represent means from about 125 such syllable reactions by a single subject (M.W.) at 16 successive character presentations and after as many reinforcements. The curved line represents the reciprocal of a fractional power of a positive growth function fitted to the data (see equation 5, p. 121). (From results published by Simley, 5.)

¹ Reinforcement in such learning is evidently secondary. (See Chapter VII.)

² This statement holds for certain conditions of training but not for all (see p. 337).

fitted curve, though in general the deviations are not excessive. The relationship thus appears to approximate rather closely the reciprocal of a positive growth function.

HABIT STRENGTH AND RESISTANCE TO EXPERIMENTAL EXTINCTION

A third way in which habit strength may manifest itself in a measurable manner is in its resistance to the effect of repeated evocations unaccompanied by reinforcement, which ordinarily produces experimental extinction (see Chapter XV). An illustration of this functional relationship is found in a study by Williams (6),

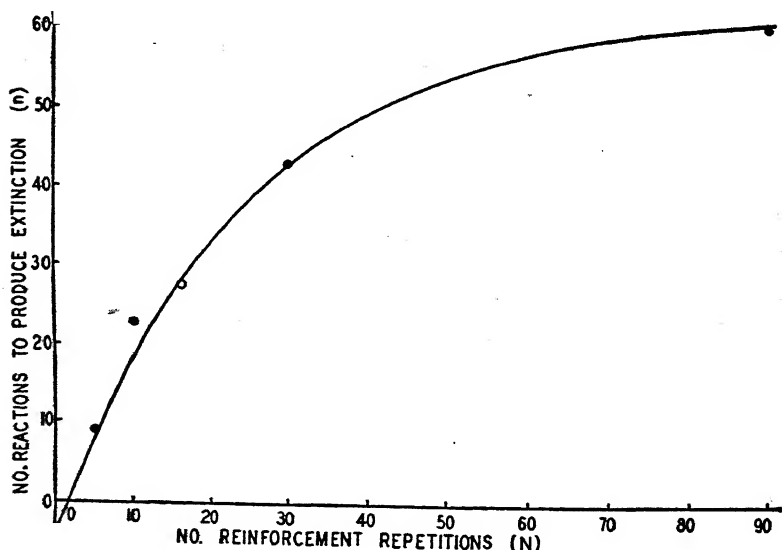


FIG. 23. Empirical learning curve plotted in terms of resistance to experimental extinction. The circles represent the mean number of unreinforced bar-pressing reactions evoked by the conditioned stimulus in different groups of hungry albino rats after varying numbers of food reinforcements. The curved line represents a positive growth function which has been fitted to the data represented by the circles. (Data from Williams, 6, and Perin; figure adapted from one published by Perin, 4.)

as supplemented by Perin (4). These investigators trained groups of hungry albino rats to depress a bar in order to secure food pellets in an apparatus resembling that of Skinner and Bugelski, described above (pp. 87, 88 ff.). The several groups of animals were given varying numbers of reinforcements, after which the reactions were

no longer followed by the food reward. The mean number of unreinforced reactions (n) which were made by the respective groups of animals before an interval of five minutes or more occurred between two successive responses was taken as the measure of resistance to extinction.¹ The relationship of these values to the number of reinforcements is shown by the circles in Figure 23. The curved line running through these data points represents a positive growth function fitted to them.

An inspection of this figure shows:

1. At zero reinforcements the number of unreinforced reactions required to produce extinction is negative. This is a quantitative expression of the fact (noted above in connection with reaction latency, p. 105) that a habit must have a certain strength before any reaction at all can be evoked (see Chapter XVIII), and therefore before any directly measurable extinction effects can possibly be observed. This negative value (-4) was obtained indirectly by extrapolating backward to zero reinforcements the function fitted to the values obtained from superthreshold strengths of the habit.

2. The greater the number of reinforcements (and, presumably, the stronger the habit), the greater will be the number of non-reinforced reactions required to produce a given degree of experimental extinction. Resistance to experimental extinction may therefore be said to be an increasing function of the number of reinforcements.

3. In general the data points, in spite of the usual deviations from the fitted curve, approximate rather closely a simple positive growth function, as we saw to be the case with reaction amplitude.

HABIT STRENGTH AND PER CENT OF CORRECT REACTION EVOCATION

A fourth way in which an increase in habit strength may manifest itself is by a change in the per cent of occurrences of the stimulating situation which evokes the reinforced reaction. This functional relationship is illustrated by the results of an unpublished experiment performed by Bertha Iutzi Hull. An albino rat was presented with a pivoted rod projecting through a cross-shaped aperture in the side of a restraining box. The hollow end of the rod was filled with sticky food. In securing this food the animal incidentally moved the rod more or less at random into all four arms of the cross, but into some much more frequently than others. At first the apparatus was set to give the rat a small pellet of

¹ An alternative and closely related measure of this same function is the time required to produce experimental extinction (t).

food whenever the rod was moved in any one of the four directions. After this had determined the relative strength of the animal's

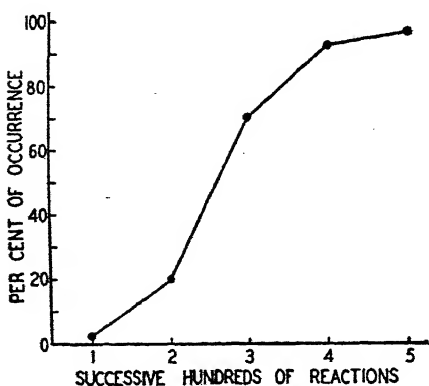


FIG. 24. Empirical learning curve plotted in terms of the per cent of correct (reinforced) reactions by an albino rat from a group of four possible directions of movement of a rod projecting through a cross-shaped aperture. (Plotted from an unpublished study by Bertha Lutzi Hull.)

reaction tendencies into each of the four arms of the cross, the end of the rod was carefully cleaned and the apparatus set to give food pellets only when the rod was moved in the hitherto least preferred direction. Owing to the large number of trials required for this bit of learning, the element of chance was largely eliminated from the results and a very smooth curve of the process was obtained from a single animal. This is shown in Figure 24. The per cent of correct reactions for successive groups of 100 trials is indicated by the circles. Since no equation

has been fitted to these data, the circles have merely been connected with straight lines.

An examination of Figure 24 shows:

1. The greater the number of trials (and, presumably, the greater the relative habit strength of the reinforced reaction), the greater will be the per cent of correct reactions.
2. The curve begins with a relatively brief period of positive acceleration.
3. The period of positive acceleration is succeeded by a relatively protracted period of negative acceleration. This had not reached the maximum of 100 per cent correct reactions when the experiment was terminated.
4. The combination of the positively and the negatively accelerated portions of the learning curve gives it a definitely sigmoid shape which is strikingly different from that of the three learning curves previously considered.

THE CONCEPT OF HABIT STRENGTH AS SUCH

We have seen exemplified four cases of relatively simple habit formation. In all these cases it has been assumed that habit

strength has progressively increased with the number of reinforcements. On this assumption, the progressive increase of habit strength in each of the four learning situations is manifested as a distinct measurable function of the number of reinforcements. While no habits manifest themselves in all of the four manners at every point of the acquisition process, most do so at one stage or another. No habits mediate overt action below the reaction threshold (p. 323 ff.). On the other hand, all well-established habits display on the presentation of the relevant stimulus a certain magnitude of reaction, a certain reaction latency, and a certain resistance to experimental extinction. Moreover, many habits at or near the reaction threshold (see Chapter XVIII), as well as all processes of selective learning at medium strengths, display a progressive increase in the frequency with which the stimulus evokes the reaction being reinforced. Indeed, certain habits, e.g., the conditioned lid reaction, may manifest themselves in all four ways at some particular stage of their formation.

From the above considerations, as well as from everyday observation, it is clear that at any time for months and years following specifiable reinforcement the presentation of the conditioned stimulus is likely to evoke the reaction. Now, it is assumed that *the immediate causes of an event must be active at the time the event begins to occur*. But at the time a habit action is evoked, the reinforcing event may be long past, i.e., it may no longer exist; and something which does not exist can scarcely be the cause of anything. Therefore, reinforcement can hardly be the direct or immediate cause of an act. We accordingly conclude that the immediate cause of habit-mediated action evocation must be a combination of (1) the stimulus event and (2) a relatively permanent condition or organization left by the reinforcement within the nervous system of the animal. This last is what is meant by the term *habit*. It will evidently be a decided convenience to speak of this persisting physical condition as distinct from either the reinforcing events which produced it or the overt activities which, on occasion, it may itself mediate.

In this connection it may be recalled from the preceding pages that the after-effects of reinforcement manifest themselves in multiple modes. From a positivistic point of view, the question naturally arises as to which of these, if any, is to be considered *the* index of habit strength. The fact seems to be that no one of them

merits this distinction more than the others. A careful survey of the evidence has led to the belief that while habit strength is the dominant factor determining the amount of each of the four aspects of reaction due to reinforcement after-effects, the latter are acting in conjunction with some other and distinct factors in each case.

What those factors may be, experimentalists are at present busily engaged in finding out, but some things are already known. It is a matter of common observation that we learn by trial and error specifically to react violently, moderately, or gently as a given situation requires in order that reinforcement shall occur (p. 304 ff.). *Thus magnitude of reaction may be learned as such.* Also, Pavlov (3) long ago showed that reactions could be conditioned to various delays by suitable methods of training. *Thus latency also may be learned as such.* Finally, whether or not a reaction will be evoked by a given stimulus apparently depends not only upon the strength of the original reinforcement but upon the other stimuli which may be acting at about the same time; upon the strength of any competing reaction tendencies associated with the other stimuli; and upon a very large number of additional factors, some of which will be elaborated in subsequent chapters (p. 341 ff.). In the calculation of the magnitudes of these various manifestations as joint functions of habit strength it will clearly be a convenience to have a single value to represent the influence of the several factors which act together to determine its amount.

It is quite possible, of course, that a theory of behavior could be developed without employing the concept of habit strength. Indeed, it is probable that all of the many constructs (such as electrons, protons, etc.) which are employed to represent unobservables in the physical sciences could be dispensed with if scientists cared to resort to the use of expressions sufficiently complex to represent explicitly all the observations from which the nature and amounts of the unobservable entities are inferred. When the relevant observations upon which the presence and magnitude of the unobservable entity depend are properly represented by a single number or sign, they can all be manipulated at once quite adequately by the manipulation of that symbol. Indeed, this is a routine practice in mathematics, where such conditions obtain. To repeat every one of them on each occasion in which the group of factors as a whole is to be manipulated would be a pedantic waste of effort. On the other hand, if the system has been properly constructed the

sign can at any time be expanded by an explicit representation of the various factors for which it stands. This manoeuvre, of course, converts the system substantially into positivistic form; which shows that fundamentally the use of constructs, where permissible (see terminal note), is no different than an ordinary positivistic procedure such as that advocated by Woodrow (7).

The use of logical constructs thus probably in all cases comes down to a matter of convenience in thinking, i.e., an economy in the manipulation of symbols. It is accordingly on the ground of convenience and economy rather than of strict necessity that the attempt is here being made to retain the substance of the common-sense notion of habit strength. Students of behavior who have a positivistic distaste for logical constructs may adapt the present systematic approach to their own preferred mode of thinking merely by recalling explicitly the various antecedent factors which determine the quantitative value of any given construct which offends them, each time it is encountered.

THE SYMBOLIC REPRESENTATION OF HABIT STRENGTH

From the foregoing it is evident that the chief advantage to be expected from the employment of the logical construct *habit strength* arises from economies in thought, i.e., in symbolic manipulation. In order to realize this advantage an appropriate symbolism must be devised. The reader will be aided in the understanding of this appropriateness if he will recall a few relevant relationships presented earlier. Specifically it will be well for him to remember that the process of reinforcement sets up a connection in the nervous system whereby an afferent receptor discharge (\dot{s}) originally involved in a reinforcement is able to initiate the efferent discharge (r) also involved in the reinforcement. But since the afferent discharge (\dot{s}) is initiated by the action of a stimulus energy (\dot{S}) on the receptor, and since the efferent discharge (r) in due course enters the effector system, producing a reaction (R), we have the sequence,

$$\dot{S} \rightarrow \dot{s} \dashrightarrow r \rightarrow R.$$

The habit organization is represented by the arrow with broken shaft between the neural processes \dot{s} and r . If we replace this arrow as a representation of habit with the more convenient and somewhat more appropriate letter H , we have the full and explicit

notation for expressing the various relationships involved in the concept of habit strength:

$$\dot{S} \rightarrow {}_sH_r \rightarrow R.$$

However, under most circumstances there is a close approximation to a one-to-one correspondence, parallelism, or constancy between \dot{S} and s on the one hand and between r and R on the other. Accordingly, for purposes of coarse molar analysis \dot{S} or s may be used interchangeably, as is the case with r and R . Since we shall be dealing with gross stimulus situations and the gross results of molar activity in the early stages of the present analysis, we shall usually employ the symbol,

$${}_sH_R.$$

Later, when we reach a point requiring a more precise and detailed analysis, it will be necessary not only to employ the notation

$${}_sH_r,$$

thus explicitly representing the neural impulse, but to distinguish through further subscript modifications various aspects of both the stimulus and the response situations. For example, \dot{S} and s represent S and s when considered as in the process of being conditioned, whereas the dots will never be used when S and s are considered as performing the function of response evocation.

HABIT STRENGTH CONCEIVED AS A FUNCTION OF THE NUMBER OF REINFORCEMENTS

Having decided to employ the construct ${}_sH_R$, we proceed at once to the problem of determining the presumptive quantitative nature of its functional relationship to its various antecedent determiners. The first of these to be considered will be the relationship of ${}_sH_R$ to the number of reinforcements (N). This type of determination presents certain difficulties.

Where the members of a functional relationship are both directly measurable, as in the Hovland reaction-amplitude study cited above, the procedure for determining the approximate mathematical relationship is fairly straightforward. A table of corresponding empirical values of the two variables is prepared and usually plotted on graph paper, as in the circle sequence of Figure 21. From an inspection of these empirical results various equations known to yield curves resembling the one shown in the graph are

fitted to the data. This consists in the main in calculating various constants¹ called for by the respective equations. Thus in the equation fitted to the Hovland data shown on page 120, the values of .141, .033, and 3.1 are all fitted, or empirical, constants; the 10 is an arbitrary value chosen for convenience because it is the base of common logarithms. In the end that equation is accepted, along with the values of the various constants associated with it, which, when the several values of one of the two variables are substituted in it, yields the closest approximation to the corresponding values of the other. In Figure 21 this approximation is shown by the nearness of the circles to the curve which was generated by the equation (see terminal notes).

When, on the other hand, one of the variables of a functional relationship under investigation is a logical construct and so is neither observable nor directly measurable, the situation is quite otherwise, and the procedure for determining the quantitative relationship is necessarily indirect and more difficult. The procedure in this case is to a considerable extent trial and error in nature, though in a rather different sense than where both sets of values are directly measurable. However, not all is trial and error, since in both situations certain supplementary principles are usually available for tentative guidance. This is notably true in the case of the probability-of-reaction-evocation curve of learning (see Chapter XVIII, p. 326 ff.).

The investigation of the functional relationship of habit strength to the number of reinforcements is so new that the greater part of the trial and error involved in its determination has yet to be performed, even though the present attempt is the second such trial to be made. Taking our point of departure from extensive observations in the field of habit formation typified by the experiments which yielded Figures 21 to 24, and profiting by the outcome of the first such attempt (2, pp. 164-165), it is concluded that very probably:

1. Habit strength is an increasing function of the number of reinforcements.

2. This function increases up to some sort of physiological limit beyond which no more increase is possible.

¹ A rather elaborate example of the determination of such constants in the fitting of a curve to empirical learning data may be found in Chapter XII, p. 200; another may be found in reference 2, pp. 103-108. At bottom, much of this is dependent in one way or another upon the use of simultaneous equations.

3. As habit strength approaches this physiological limit with continued reinforcements the increment ($\Delta_s H_R$) resulting from each additional reinforcement decreases progressively in magnitude.

Now, there are numerous algebraic expressions which yield results conforming to the above specifications. One of these, however, has a rather special promise because it is known to approximate closely a very large number of observable empirical relationships in all sorts of biological situations involving growth and decay. Indeed, Figures 21, 22, and 23 are all cases in point. The basic principle of the simple positive growth function (Figures 21 and 23) is that *the amount of growth resulting from each unit of growth opportunity will increase the amount of whatever is growing by a constant fraction of the growth potentiality as yet unrealized.*

THEORETICAL CURVE OF HABIT-STRENGTH GROWTH EXEMPLIFIED

The characteristics of the positive growth function may be exhibited by means of an example. From the foregoing it is evident that the rate of habit growth is dependent upon three factors or *parameters*:

1. The physiological limit or maximum (M)
2. The ordinal number (N) of the reinforcement producing a given increment to the habit strength ($\Delta_s H_R$)
3. The constant factor (F) according to which a portion ($\Delta_s H_R$) of the unrealized potentiality is transferred to the actual habit strength at a given reinforcement

There must also be devised a unit in which to express habit strength. This is taken arbitrarily as 1 per cent of the physiological maximum (M) of habit strength attainable by a standard organism under optimal conditions. In order to make the name of the unit easy to remember, it will be called the *hab*,¹ a shortened form of the word *habit*. Thus under the conditions stated above there would be 100 habit units, or habs, between zero and the physiological limit, i.e., one hab = $\frac{M}{100}$.

We proceed now with our example. Suppose that the growth constant (F) in a given reinforcement situation is taken as 1/10.

¹ Pronounced *hăb*, as in *cab*.

One-tenth of the total possibility of learning (100 units) is 10 habs (1/10 of 100 = 10). The generation of 10 units of habit strength from a base, zero, leaves 100 — 10, or 90 units of growth yet possible of realization. Consequently the habit increment resulting from the second reinforcement must be 1/10 of 90, or 9; i.e., the second $\Delta_s H_R = 9$ habs. Subtracting 9 from 90, we have left 81 units of possible growth. One-tenth of 81 in turn yields our next $\Delta_s H_R$ of 8.1 habs; and so on. This process can be repeated as many times as there are successive repetitions of the reinforcement. Column 2 of Table 1 shows the first 30 successive $\Delta_s H_R$'s computed in this way. These $\Delta_s H_R$'s are shown graphically at the left

TABLE 1

ANALYTICAL TABLE SHOWING THE THEORETICAL EVOLUTION OF A TYPICAL "GROWTH" FUNCTION IN WHICH EACH INCREMENT TO THE HABIT IS 1/10 OF THE POTENTIAL HABIT STRENGTH AS YET UNFORMED. (See text for details and Figures 25 and 26 for graphical representation.)

Ordinal Number of Reinforcements	Increment of Habit ($\Delta_s H_R$)	Total Accumulated Habit in Hab Units ($\Sigma \Delta_s H_R$)
1	10	10
2	9	19
3	8.1	27.1
4	7.29	34.39
5	6.561	40.951
6	5.9049	46.856
7	5.3144	52.1703
8	4.7830	56.9533
9	4.3047	61.2580
10	3.8742	65.1322
11	3.4868	68.6189
12	3.1381	71.7570
13	2.8243	74.5813
14	2.5419	77.1232
15	2.2877	79.4109
16	2.0590	81.4698
17	1.8530	83.3228
18	1.6677	84.9905
19	1.5009	86.4915
20	1.3509	87.8423
21	1.2158	89.0581
22	1.0942	90.1523
23	.9848	91.1371
24	.8863	92.0234
25	.7977	92.8210
26	.7179	93.5389
27	.6461	94.1850
28	.5815	94.7665
29	.5233	95.2899
30	.4710	95.7609

edge of Figure 25, piled one upon the other in the order in which they were derived. It is notable that these increments become smaller and smaller until, with very large values of N , they become infinitesimal.

In column 3 of Table 1 are presented the cumulative values of column 2. These latter values, in their turn, are represented graphically in the main portion of Figure 25. The contour of this columnar figure is a rather precise representation of what is here conceived to be the basic "curve of learning" from which all other theoretical curves of learning are derived in one way or another.

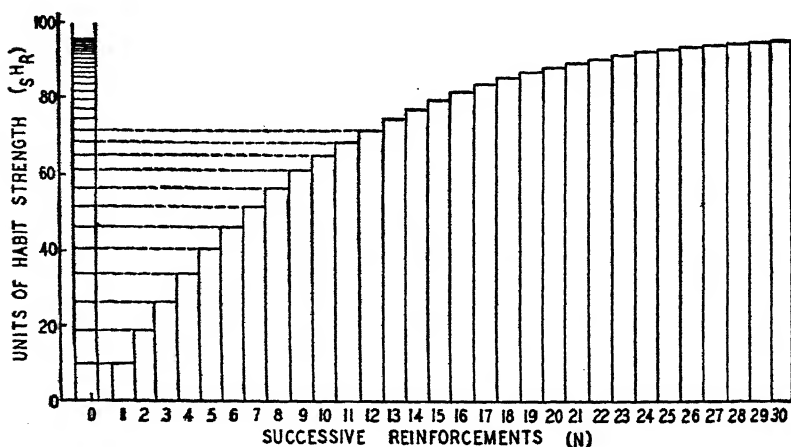


FIG. 25. Diagrammatic representation of a theoretical simple positive growth function. At the left are given the successive increments of habit accretion for successive reinforcements as shown in column 2 of Table 1. At the right may be seen the amount of accumulated habit strength at the successive reinforcements as shown in column 3 of Table 1.

It will be noticed that it rises at first with comparative rapidity, the rate of rise gradually diminishing until at high values of N it becomes practically horizontal. Because of their progressively diminishing rate of rise such curves are said to be negatively accelerated.

The notched appearance of the contour of the main portion of Figure 25 is due to the fact that each reinforcement is a unit, i.e., is essentially indivisible into fractional parts such as halves or thirds of a reinforcement. The usual method of plotting learning functions by smooth-line curves running through the points representing the readings taken after each reinforcement, is shown for

the values in column 3 of Table 1 in Figure 26. It is to be noted, however, that while for many purposes this method of representing the course of learning is to be preferred because of its convenience, there is danger of its giving the uninitiated a false impression of smooth continuity. Such a smooth, continuous process could result

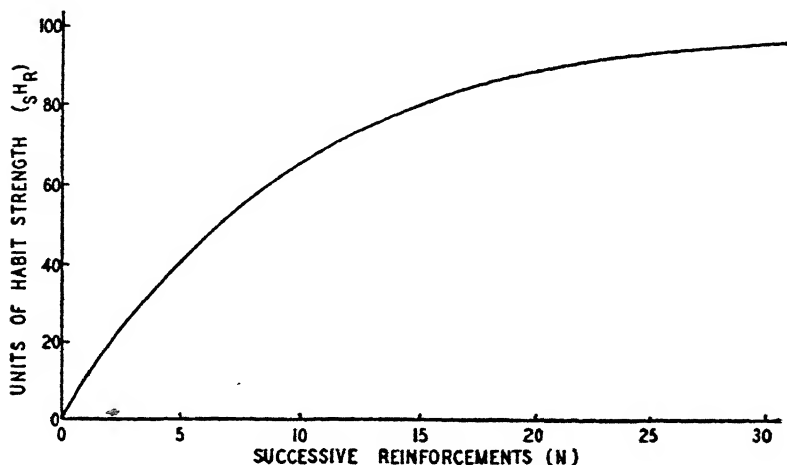


FIG. 26. Theoretical curve of learning a simple conditioned reaction (sH_R) as a function of the number of reinforcements (N), plotted in the customary manner, which implicitly but falsely assumes that repetitions can be subdivided indefinitely (see text).

only if the successive repetitions of reinforcement were to be indefinitely subdivided into fractional parts. The cyclical nature of the reinforcement process precludes this.

SUMMARY

The effect of reinforcement may become manifest in overt action upon the presentation of the associated stimulus at any time during the subsequent life of the organism. This central fact shows conclusively that reinforcement leaves within the organism a relatively permanent connection between the receptor and the effector associated in the original reinforcement. It is this which in the present system is meant by the term "habit," a technical adaptation of the common-sense concept that goes by the same name.

Since the organization of the nervous system upon which habitual action is evidently based lies deeply hidden and quite remote

from any immediate means of direct observation, habit has the status of an unobservable, i.e., it is a logical construct. As such it is prevented from becoming a metaphysical entity by being firmly anchored, both antecedently and consequently, to phenomena which alike are observable and measurable. On the antecedent or causal side, habit is known to be dependent upon various factors associated with reinforcement, but notably upon the number of reinforcements. On the consequent or effect side, habit manifests itself in action, ideally on the presentation of the stimulus aggregate originally associated with it at its reinforcement. It is important to note, however, that between ${}_sH_R$ and observable response phenomena there intervene several additional symbolic constructs (see Figure 84), each of which is directly or indirectly anchored both antecedently and consequently to quantitatively observable phenomena. The strength of the habit is manifested indirectly by various measurable aspects of action: (1) reaction amplitude or magnitude (A), (2) reaction latency (${}_st_R$), (3) resistance to experimental extinction (n), and (4) probability (p) of occurrence, i.e., per cent of appropriate stimulations which evoke the associated reaction (p. 326 ff.).

From a study of the empirical relationships of the number of reinforcements to typical examples of each of the four forms of habit action it is concluded that while all are dependent in the main upon habit strength, each is also dependent in part, and differentially so, upon other factors which enter the reinforcement situation. For this reason it will be convenient to have a representation of habit strength independent of any of its potential behavioral manifestations.

The determination of the functional relationship of an observable to an unobservable presents a rather different and more difficult problem than that of two observables. The two determinations are alike in that they are both dependent partly upon a process of trial and error, though each in a somewhat different sense. In the case of an observable and an unobservable the relationship which is most plausible in the light of all related observable phenomena is postulated. This postulated relationship is then employed in all appropriate deductive situations. If the assumption is false such deductions will lead, sooner or later, to inconsistencies with observation and so to its correction. On the other hand, if a very large number of such deductions uniformly agree with obser-

vation, this will indicate that the postulated relationship is valid to an increasing degree of probability.

The postulated relationship of habit strength to the number of reinforcements is that each reinforcement results in the addition of an increment to the habit strength ($\Delta_s H_R$) which is a constant fraction (F) of the difference between the physiological maximum (M) of habit strength and the habit strength immediately preceding the reinforcement. This is a relatively uncomplicated mathematical relationship which we shall call a *simple positive growth function*.

NOTES

The Present Trial-and-Error Status of Our Hypothesis as to the Relation of Habit Strength to Number of Reinforcements

On page 113 it was pointed out that the determination of the correct functional relationship of an unobservable to an observable is to a considerable extent dependent upon trial and error. As a matter of fact, the formulation of this relationship contained in the present chapter is the second such trial. The first such formulation made up a part of a postulate set upon which was based a highly formalized theory of rote learning (2). The assumption in that case was that habit strength is directly proportional to the number of reinforcements up to the physiological limit. That postulate generated a theorem which is clearly contrary to fact (2, pp. 164-165). The present formulation corrects the defect thus revealed and so is presumably a closer approximation to the truth than was the first attempt; therefore it may be expected to survive somewhat longer.

How to Compute Habit Strength

The rather clumsy method of generating the theoretical learning curve used above for illustrative purposes (Table 1 and Figures 25 and 26) was chosen for expository reasons because of its psychological simplicity. For systematic purposes the outcome of this arithmetical procedure as shown in column 3 of Table 1 is usually represented by an equation. It may be shown by rather simple mathematical procedures that column 3 ($\Sigma \Delta_s H_R$) as a function of the number of repetitions (N) is given by the equation:

$$\sum_s H_R = M - Me^{-iN} \quad (1)$$

where $M = 100$, N is the number of reinforcement repetitions, e is 10, and

$$i = \log \frac{1}{1-F} \quad (2)$$

where F is the reduction constant, in the above example taken as 1/10, i.e., $F = .1$. Accordingly,

$$i = \log \frac{1}{1-.1} = \log \frac{1}{.9} = \log 1.111111+$$

Now, by ordinary logarithm tables,

$$\log 1.111111+ = .04574 \text{ (approximately).}$$

Therefore, if we wish to determine the amount of sH_R after five reinforcements we have,

$$\begin{aligned} {}^5sH_R &= 100 - \frac{100}{10^{.04574 \times 5}} \\ &= 100 - \frac{100}{10^{.2287}} \\ &= 100 - \frac{100}{1.6932} \\ &= 100 - 59.0597 \\ &= 40.94, \end{aligned}$$

which, except for the facts that logarithm tables give only approximate values and that decimals have been dropped, would agree exactly with the corresponding values in Table 1.

How to Compute Increment of Habit Strength per Single Reinforcement

In a similar manner the value of the increment in habit strength due to one reinforcement (ΔsH_R) at any particular stage of the learning is given by the equation,

$$\Delta sH_R = M - x - (M - x) 10^{-i} \quad (3)$$

where x is the strength of the habit immediately preceding the reinforcement which produces the increment, and M , i , etc., have the same values as above. For example, the value of ΔsH_R which would result from a sixth reinforcement following the fifth reinforcement (the sH_R of which was calculated above) is calculated as follows: substituting in equation 3, we have,

$$\begin{aligned} \Delta sH_R &= 100 - 40.94 - (100 - 40.94)10^{-.04574} \\ &= 59.06 - 59.06 \\ &= 59.06 - \frac{59.06}{1.11111111} \\ &= 59.09 - 53.154 \\ &= 5.906. \end{aligned}$$

The value 5.906 is as close an approximation to the value in column 2 of Table 1, where $N = 6$, as is to be expected from the approximations attainable where ordinary logarithm tables are employed in the computations.

Equations Fitted to the Learning Curves

We turn now to the details of the analysis of the data of Figures 21, 22, and 23. The Hovland data represented in Figure 21 are fitted fairly well by the equation,

$$A = 14.1(1 - 10^{-.033N}) + 3.1, \quad (4)$$

from which the curve running through the data points of Figure 21 has been plotted. This is a simple positive growth function.

The Simley data, represented in Figure 22, are fitted fairly well by the equation,

$$s'_R = \frac{5.11}{[100(1 - 10^{-.122 N})]^{-.4}} \quad (5)$$

from which the curve running through the data points of Figure 22 was plotted, where s'_R represents the time (t) from the beginning of S to the beginning of R , i.e., the reaction latency. The equation represents the reciprocal of a slightly complicated positive growth function. Since the denominator of the right-hand member of the equation becomes zero when $N = 0$, s'_R becomes infinite under the same conditions, i.e., no reaction whatever occurs when there is no habit strength. In point of fact, no reaction occurs when the effective reaction potential is less than the reaction threshold (see Chapter XX).

The Williams data represented in Figure 23 are fairly well expressed by the equation,

$$n = 66(1 - 10^{-.018 N}) - 4, \quad (6)$$

from which the curve drawn through the data points of Figure 23 was plotted, where n represents the number of unreinforced reactions performed before a given degree of experimental extinction develops.

The Relation of the Equation Expressing Habit Strength (1) to Equations 4, 5, and 6

It is implicit in the foregoing analysis that equations 4, 5, and 6, and the data represented in Figure 24, are composites, one component of which in each case takes the general form of equation 1. For example, equation 4,

$$A = 14.1(1 - 10^{-.033 N}) + 3.1,$$

when thus analyzed breaks up into the following:

$$sH_R = 100 - 100 \times 10^{-.033 N} \quad (7)$$

and

$$A = .141 \times sH_R + 3.1. \quad (8)$$

Of these, equation 7 expresses habit strength (sH_R) as a function of the number of reinforcements, and equation 8 expresses the amplitude of the reaction as a function of sH_R , the latter relationship being linear (see Figure 87).

The equation from the analysis of sH_R as a function of N which emerges from equation 5 (Figure 22) is,

$$sH_R = 100 - 100 \times 10^{-.382 N}, \quad (9)$$

and that emerging from the analysis of equation 6 (Figure 23) is,

$$sH_R = 100 - 100 \times 10^{-.018 N}. \quad (10)$$

It will be observed that the general form of equations 7, 9, and 10 is the same, though there is a wide variation in the coefficient of N .

The various equations corresponding to equation 8 represent the joint relationship of the final effector process to (1) the results of previous learning retained in the nervous system and (2) the particular stimulus conditions existent at the time of action evocation. Since numerous factors other than the mere sH_R enter into the ultimate evocation process, the various functional relationships

corresponding to equation 8 cannot properly be taken up until these factors have been examined. Accordingly, the final analysis of Figures 21, 22, 23, and 24 will be delayed until Chapter XVIII.

Does sH_R Qualify as a Quantitative Scientific Construct?

Even though it be granted that the nature and quantitative aspects of habit action are immediately and necessarily dependent upon the state of the nervous system, it does not follow that this state, as represented by sH_R , qualifies as a satisfactory scientific construct. As already pointed out in the text, a typical scientific construct represents the joint, *unitary* action of a number of independent directly measurable variables in the determination of some subsequent event. *If these variables do not act as a unit in a given situation they cannot properly be treated as a quantitative construct in that situation.*

For example, there are excellent empirical grounds for believing that habit strength is dependent not only upon the number of reinforcements, but upon a number of other measurable antecedent conditions under which reinforcement occurs. These antecedent factors play the rôle of independent variables. When finally worked out, the quantitative value of sH_R is to be thought of as a dependent variable whose value may be calculated by substituting in a more or less complex equation or formula the values of numerous independent variables such as the number of reinforcements, the magnitude of the reinforcing agent employed, the time from the onset of the conditioned stimulus to the reaction, the time from the reaction to the reinforcement, the number and nature of the irrelevant stimuli present during the reinforcement, and so on. All of these independent variables may be assumed to be related to habit strength in different ways, some favoring it and others hindering it in varying degrees. Now, in such a situation it is evident that an increase or decrease in the value of one of these independent variables may exactly offset a certain amount of decrease or increase in the value of any of the others. This means that a given amount of habit may be produced indifferently by innumerable combinations of the antecedent variable values. It means, further, that *all of these different combinations of antecedent reinforcement variables will yield, other factors equal, in the action evocation situation exactly the same amplitude of reaction, latency of reaction, persistence of reaction, and probability of reaction.* Fortunately these implications of the unitary nature of a truly scientific construct are capable of fairly straightforward empirical test.

It is evident that whether sH_R , or indeed any other behavioral construct, will satisfy the requirements just outlined depends upon the outcome of a great amount of precise quantitative experimentation, only a little of which has yet been performed. This uncertainty applies not only to the constructs employed in the present system but to those of all other theoretical approaches, including the potential systems of the various Gestalt schools. If the ultimate verdict proves to be affirmative, the task of the behavior sciences will be far simpler than if it turns out to be negative. Meanwhile we must resolutely face the realities of the situation, whatever the future holds for us. It is believed that the quickest and most economical way to discover whether or not behavior is so constituted as to permit the use of genuine scientific constructs is boldly to postulate it, deduce the implications of this assumption in all possible situations, and then accept or reject the hypothesis as the deductions agree or disagree with

empirical findings. The beginning of such an attempt is being made in the present work.

REFERENCES

1. HOVLAND, C. I. The generalization of conditioned responses. IV. The effects of varying amounts of reinforcement upon the degree of generalization of conditioned responses. *J. Exper. Psychol.*, 1937, 21, 261-276.
2. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
3. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
4. PERIN, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. *J. Exper. Psychol.*, 1942, 30, 93-113.
5. SIMLEY, O. A. The relation of subliminal to supraliminal learning. *Arch. of Psychol.*, 1933, No. 146.
6. WILLIAMS, S. B. Resistance to extinction as a function of the number of reinforcements. *J. Exper. Psychol.*, 1938, 23, 506-521.
7. WOODBOW, H. The problem of general quantitative laws in psychology. *Psychol. Bull.*, 1942, 39, 1-27.

CHAPTER IX

Habit Strength as a Function of the Nature and Amount of the Reinforcing Agent

We have seen that if a habit is to be set up, the act involved must be associated either with a need reduction or with some stimulus which has itself been associated with a need reduction. Now, the amount or quality of the reinforcing agent at every reinforcement may clearly vary in such a way that the degree of need reduction will range from very large amounts through very small amounts down to a value of zero, at which point presumably no reinforcement at all will occur. In short, the amount (or quality) of the agent employed at each reinforcement appears as the second of the numerous antecedent conditions determining habit strength.

It is evident even from the preceding analysis that somewhere between the extremes of zero need reduction and a maximum value of such reduction a transition must be made from zero amount of the reinforcing agent to an amount of considerable magnitude. The question arises: Is this transition abrupt, or is it gradual and progressive? And if it is progressive, what is the law of its progress? A small amount of experimental evidence bearing on this question is available, some of it concerned with conditioned-reflex learning and some with selective learning.

THE LIMIT OF CONDITIONED-REFLEX LEARNING AS A FUNCTION OF THE AMOUNT OF THE REINFORCING AGENT

Gantt (2) has reported a conditioned-reflex experiment of the Pavlovian type in which each of several animals was conditioned to four different stimuli, one stimulus being reinforced by one-half gram of food, one by one gram, one by two grams, and one by twelve grams. The four conditioned reactions were reinforced in random order not only on different days but during the experimental session on the same day. After considerable amounts of training it was found that some dogs developed clearly differentiated reactions to the several stimuli, though others were unable to do this. The mean results from one of the former, named "Billy," a very stable animal, are shown by the circles in Figure 27. The

curve running through these values is a simple positive growth function originally fitted to them by Dr. Gantt (2). The closeness of the approximation of the fitted to the empirical values indicates considerable consistency despite the small number of points involved.

Notwithstanding the resemblance of this curve to those characteristic of ordinary learning, it is not to be confused with a

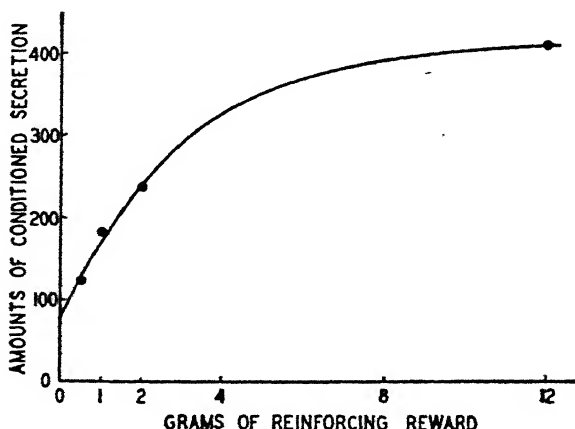


FIG. 27. Graphic representation of the empirical functional relationship between the amount of the reinforcing agent (food) employed at each reinforcement of four conditioned reactions to as many different stimuli, and the final mean amount of salivary secretion evoked by each stimulus at the limit of training. The appreciable secretional value of 75 units when the fitted curve is extrapolated to where the amount of reinforcing agent equals zero is presumably due to secretion evoked by static stimuli arising from the experimental environment. Plotted from unpublished data from the dog "Billy," kindly furnished by Gantt (2) and here published with his permission. The experimental work upon which this graph is based was performed previous to 1936 (personal communication from Dr. Gantt).

learning curve; on the contrary, each of the data points of this investigation represents the mean response on the part of the dog at the limit of training to the respective amounts of reinforcement; i.e., *each represents the final horizontal portion or asymptote of a separate and distinct curve of learning.*

THE RATE OF SELECTIVE LEARNING AS A FUNCTION OF THE AMOUNT OF THE REINFORCING AGENT

Grindley (3) has reported a study which is closely analogous to that of Gantt but which involves selective rather than condi-

tioned-reflex learning. This investigator trained five groups of twelve-day-old chicks to traverse a runway eight inches wide and four feet long. The reinforcing agent or reward was grains of boiled rice placed in a shallow tray at the end of the runway. One group of chicks found and ate one grain of rice on reaching the tray after successfully traversing the runway; one group received two grains; another, four grains; and a fourth, six grains. A fifth group received no food whatever on reaching the tray. The score adopted as an index of learning was $\frac{100}{t}$, i.e., 100 times the reciprocal of the time in seconds required by a chick to traverse the runway and begin eating the rice.

Grindley published composite learning curves for his several groups of chicks. Pooled measurements of the comparable later

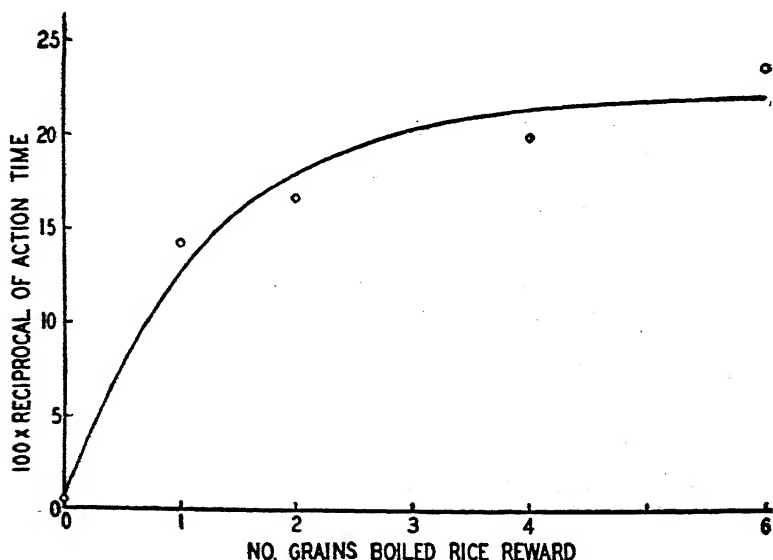


FIG. 28. Empirical graph representing the rate of selective learning as a function of the magnitude of the reinforcing reward. Each circle represents the mean score at the last five of seven trials of ten chicks in traversing a four-foot runway to secure differing numbers of boiled-rice grains. (Derived from measurements made of learning curves published by Grindley, 3, p. 174.)

portions of these several curves are shown in Figure 28. In order to permit further comparison with the Gantt study, a simple positive growth function has been fitted to the Grindley data; this is

represented by the curve passing among the circles of Figure 28. While the deviations of the data points from the curve are considerable, the fit is still close enough to indicate a fair approximation.

Grindley's results are corroborated and extended by a study reported by Wolfe and Kaplon (4). These investigators repeated the substance of Grindley's experiment, utilizing with groups of chicks an explicit trial-and-error task, that of learning a simple T-maze. A comparison was made between the reinforcing power of one-fourth of a kernel of popcorn, a whole kernel, and four quarters of a kernel given at once. It was found that a whole kernel was more reinforcing than a quarter of a kernel, which confirms the findings of both Gantt and Grindley. However, four quarter kernels given at once proved to be distinctly more reinforcing than did a single intact kernel. In the case of the four quarter kernels, of course, the chickens pecked and swallowed four times after each successful act. We thus appear to have in this case the paradox of four more or less distinct reinforcements contributing by summation to produce the increment of learning resulting from a single successful action sequence.

In spite of the great difference in the organisms involved, in the index of learning employed, and in the stage of learning represented, the results of the selective learning experiments show a striking general agreement with the conditioned-reflex results of Gantt. It accordingly seems fairly clear from the two types of studies that the rate of learning is an increasing monotonic function of the amount of the agent employed at each reinforcement.

HOW DOES THE AMOUNT OF THE REINFORCING AGENT INFLUENCE THE TWO PARAMETERS OF THE CURVE OF HABIT FORMATION?

We saw above (Chapter VIII) that the curve of habit strength as a function of the number of reinforcements was dependent upon two constants, (1) the physiological maximum of habit strength (M), and (2) the fractional part (F) of the as yet unrealized potentiality of habit-strength acquisition, which is added to the actual habit strength at each reinforcement. Assuming the soundness of this hypothesis, it is evident that the influence of increasing the amount of the reinforcing agent on the size of the increment of habit strength ($\Delta_s H_R$) at a given reinforcement must result from an increase in one, and possibly both, of these parameters.

The parameter, or parameters, involved could be determined if we had reliable learning curves which were carried up to, or near, the limit of practice under different amounts per reinforcement of the same reinforcing agent. Unfortunately no such investigation has yet been reported in detail, though there are indications in both the study of Gantt and in that of Wolfe and Kaplon that, at the limit of practice, habit strength is definitely greater where the amount of the agent employed in reinforcement is greater. Gantt has published no practice curves, and those published by Wolfe and Kaplon are not sufficiently regular to make profitable a precise analysis from this point of view. An inspection of these latter curves and the tables accompanying them suggests, however, that in habit formation the F -value may be approximately constant for different amounts of the reinforcing agent or reward. The only possible remaining parameter which could produce the slower learning with small amounts of the reinforcing agent is the asymptote or upper limit of the learning curve.

In this connection it will be recalled from the last chapter that the physiological limit of habit strength under absolutely optimal conditions was taken as 100 habs, p. 114. To this value was assigned the symbol M . The introduction of the presumption that the asymptotes of learning curves may vary below this level, depending on the amount and quality of the reinforcing agent, makes it necessary to employ a separate symbol (M') to represent such limits or asymptotes. We now state the working hypothesis at which we have arrived: *In a learning situation which is optimal in all other respects, the limit (M') of habit strength (sH_R) attainable with unlimited number of reinforcements is a positive growth function of the magnitude of the agent employed in the reinforcement process.* This tentative conclusion is based on admittedly inadequate grounds and will therefore be subject to reëxamination and revision when more satisfactory evidence becomes available.

An important extension of this hypothesis at once suggests itself. Clearly a reinforcing agent may vary in quality as well as in quantity. More specifically, with quantity remaining constant, a reinforcement by one agent may reduce the need more than a reinforcement by another. For example, a standard food may be adulterated by adding varying amounts of some inert and tasteless substances such as a flour consisting of ground wood. It is evident that two grams of a standard dog ration mixed with 50 per cent wood flour would reduce an animal's food need only half as much

as would the same quality of the food unadulterated. While no report of such an experiment has been found, it seems reasonable to suppose on the analogy of the experiments of Gantt and Grindley that a food so adulterated would be less effective as a "primary" reinforcing agent than would an equal weight of the natural food. However, judging from the shape of the curve of Figures 27 and 28 it is to be expected that the limit of learning (m) resulting from reinforcement with the adulterated agent would be more than half as effective as would be that resulting from the use of the unadulterated agent.

We conclude, then, that *habit strength at the limit of practice (m) will vary with the quality, as well as the quantity, of the reinforcing agent from a minimum of zero to a physiological maximum of 100 habs, and that the rate of approach to that limit (F) will remain unchanged.*

SOME IMPLICATIONS OF THE AMOUNT-OF-REINFORCEMENT HYPOTHESIS

The meaning of the working hypothesis just formulated may be clarified by indicating one or two of its implications. Let it be supposed, on the analogy of the Gantt experiment, that the maximum amount of a given food when used as a reinforcing agent would yield at the limit of practice a habit strength of 80 habs. Calculations based on the F -constant fitted to the Gantt data show that if, under these conditions, one gram of this food were used at each reinforcement, the maximum habit strength to be expected at the limit of practice would be 23.75 habs. A parallel computation shows that the maximum to be expected from the use of six grams of this food at each reinforcement would be 70.14 habs.

With these maxima available it is possible to calculate the theoretical course of habit-strength acquisition under the respective conditions by substituting first one of the values for m in the simple positive growth function, and then the other, letting the fractional incremental factor, F , equal $1/10$, as in the illustration of Chapter VIII. In this way were computed the values from which were plotted the two main curves of Figure 29. These curves, taken together, show the effect upon the course of habit formation implied by the hypothesis put forward above.

An additional implication of the working hypothesis may still further clarify its meaning. Let it be supposed that the habit

has been reinforced with one gram of the food fifteen times and that the reinforcement is then suddenly shifted to six grams on the next fifteen reinforcements. Neglecting the presumptive perseverating influence of secondary reinforcement in the situation, the outcome is easily calculated by methods analogous to the determination of the two main curves of Figure 29. This is shown by the dotted curve rising from the one-gram curve at the fifteenth rein-

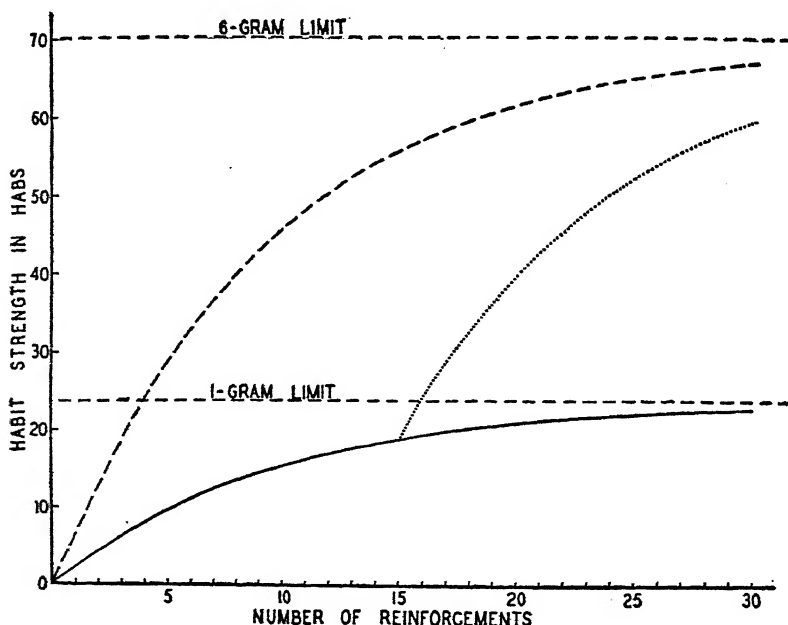


FIG. 29. Graphic representation of the theoretical course of habit-strength acquisition with a six-gram food reinforcement (broken line) and with a one-gram food reinforcement (solid line). The dotted curve indicates the theoretical course of habit-strength acquisition on the assumption that reinforcement is abruptly shifted to six grams on the sixteenth trial of the one-gram reinforcement curve.

forcement. A glance at Figure 29 shows that according to the present hypothesis an increase in the amount of the reinforcing agent should be followed by a marked increase in the rate of habit-strength acquisition; this, however, would gradually lessen as the new limit is approached, in accordance with the nature of the simple growth function. In concluding our discussion of Figure 29 it is to be noted that exactly analogous curves would also be produced by suitable variations in the quality of the reinforcing agent.

Finally, an additional case may be mentioned—that in which the six-gram habit would be reinforced ten times. This would generate a habit strength of 45.68 habs, which is considerably above 23.75 habs, the maximum attainable by means of a one-gram reinforcement. Then the reward would suddenly be shifted to one gram. Assuming these conditions, and ignoring secondary reinforcing effects, it is to be expected that successive reinforcements would result in a progressive weakening of the habit. Consideration of this interesting but complex problem must be deferred until the phenomena of experimental extinction have been taken up in detail (p. 258 ff.).

THE AMOUNT OF THE REINFORCING AGENT AND THE PROBLEM OF INCENTIVE

Although the systematic presentation of the subject of motivation has been reserved for a later chapter (p. 226 ff.), it becomes desirable here to touch briefly on one of its phases. Motivation has two aspects, (1) that of drive (*D*, or *S_D*) characteristic of primary needs, and (2) that of incentive. The amount-of-reinforcement hypothesis is closely related to the second of these aspects. The concept of incentive in behavior theory corresponds roughly to the common-sense notion of reward. More technically, the incentive is that substance or commodity in the environment which satisfies a need, i.e., which reduces a drive.

Let us suppose that in a simple selective learning situation involving a hunger drive, the food employed as a reinforcing agent is plainly visible at the moment the organism performs the several acts originally evoked by the stimulus situation; that the individual reinforcements are separated by a number of hours; and that the amount of food employed in the several reinforcements varies at random from almost zero to very large amounts. Under such circumstances it follows from the principle of reinforcement that the visual stimulus arising from the food will be conditioned to the successful act by the subsequent reinforcing state of affairs, the consumption and absorption of the food. Moreover, in case the amount of food shrinks to zero there will be no direct reinforcement at all. From these considerations, coupled with the amount-of-reinforcement hypothesis, it may be inferred that the successful reaction will be more strongly conditioned to the stimulus aggregate arising from a large piece of food than to that from a small one.

Therefore, given a normal hunger drive, the organism will execute the correct one of several acts originally evoked by the situation more promptly, more vigorously, more certainly, and more persistently when a large amount of food is stimulating its receptors than when they are stimulated by a small amount.

Substantial confirmation of this deduction is furnished by a recent experiment reported by Fletcher (1), who trained a chimpanzee to secure pieces of banana by pulling into its cage a weighted car by means of an attached rope. It was found that the animal would perform more work for a large piece of banana than for a small one. The maximum amount of work which would be performed for a given amount of banana incentive between the range of .64 and 3.77 units was practically linear.

An extrapolation of Fletcher's linear relationship just mentioned suggests that the animals would have performed with a zero amount of incentive more than half as much work as was performed with the incentive at 3.77 units. This might well occur on a few occasions due to the conditioning of the reaction to the stimuli arising from the apparatus and other environmental elements. On the other hand, in case no food is present the strength of the remaining stimulus elements conditioned to the reaction may be so far depleted by the absence of the incentive component of the stimulus compound (see Chapter XV) that the effective habit strength will either be less than the reaction threshold or than the strength of some competing reaction tendency evoked by the stimulus situation; in either case the reinforced reaction may not occur at all. In the event that it does occur under such conditions, however, experimental extinction will presently set in and soon terminate it.

SUMMARY

Since the amount of need reduction presumably varies with the amount of the reinforcing agent consumed by the organism, it follows as a strong probability from the dependence of reinforcement upon the amount of need reduction that the increment of habit strength ($\Delta_s H_R$) per reinforcement will be an increasing function of the amount of the reinforcing agent employed. This *a priori* expectation is substantiated by empirical investigations involving both selective and conditioned-reflex learning. Moreover, these studies indicate that the relationship is that of a simple positive growth function.

Because the law of simple habit acquisition is presumably a positive growth function of the number of reinforcements, it follows that the increased rate of habit acquisition with increased amounts of the reinforcing agent employed may be due to one or both of two factors: (1) an increased limit of potential habit-strength growth, or (2) an increased fraction of this potentiality which is added to the habit at each reinforcement. The empirical evidence on this point is at present inadequate for final decision. Pending the appearance of more complete evidence, the working hypothesis is adopted that an increase in either the quality or the quantity of a reinforcing agent increases the rate of learning by raising the limit (m) to which the curve of habit strength approaches as an asymptote, the rate of approach (F) to this limit possibly remaining constant for all qualities and amounts of the reinforcing agent employed.

From the amount-of-reinforcement hypothesis may be derived a special case of one phase of motivation, that of incentive or secondary motivation. This is the situation where the incentive (reinforcing agent) contributes a prominent, direct component of the stimulus complex which is conditioned to the act being reinforced. The stimulus component arising from a large amount of this substance will be different from that arising from a small amount, and will differ still more from a stimulus situation containing a zero amount. It follows from this and the amount-of-reinforcement hypothesis that in the course of reinforcement by differing amounts of the reinforcing agent, the organism will inevitably build up stronger reaction tendencies to the stimulus arising from large amounts than to that from small amounts, and no habit strength at all will be generated by zero amounts. It thus comes about, primary motivation (e.g., hunger) remaining constant, that large amounts of the agent will evoke more rapid, more vigorous, more persistent, and more certain reactions than will small or zero amounts. Thus a reinforcing agent as a stimulus becomes an incentive to action, and large amounts of the agent become more of an incentive than small amounts. This *a priori* expectation is well substantiated by quantitative experiment as well as by general observation.

NOTES

The Equations Which Were Fitted to Gantt's and Grindley's Empirical Data

The equation fitted by Gantt (2) to the data represented by the circles shown in Figure 27 is,

$$A = 335 (1 - 10^{-.153 w}) + 75, \quad (11)$$

where A is the amount of salivary secretion in arbitrary units during a constant number of seconds, and w is the weight of the reinforcing agent in grams employed at each trial. The $+ 75$ is arrived at by extrapolation.

The equation fitted to the Grindley data represented by the circles of Figure 28 is,

$$\frac{100}{t} = 21.38 (1 - 10^{-.362 n'}) + .5,$$

where t is the time in seconds required to traverse the four-foot runway to the food and n (number of rice grains) is the magnitude of the reinforcing agent employed. The $+ .5$ represents the score resulting from spontaneous exploratory activity previous to receiving reinforcement at the end of the runway.

The Equations From Which the Curves of Figure 29 Were Derived

The equation from which the upper curve of Figure 29 was derived is,

$$sH_R = 70.14(1 - 10^{-.04576 N}),$$

in which the 70.14 was derived from the equation,

$$M' = 80(1 - 10^{-.153 w}), \quad (12)$$

where $w = 6$ grams.

The equation from which the lower curve of Figure 29 was derived is,

$$sH_R = 23.75(1 - 10^{-.04576 N}),$$

in which the 23.75 was derived from the equation,

$$M' = 80(1 - 10^{-.153 w}),$$

where $w = 1$ gram.

The equation from which the dotted curve rising from the 1-gram curve was derived is,

$$sH_R = (70.14 - 18.86)(1 - 10^{-.04576 N}) + 18.86.$$

REFERENCES

1. FLETCHER, F. M. Effects of quantitative variation of food-incentive on the performance of physical work by chimpanzees. *Comp. Psychol. Monogr.*, 1940, 16, No. 82.
2. GANTT, W. H. The nervous secretion of saliva: The relation of the conditioned reflex to the intensity of the unconditioned stimulus. *Proc. Amer. Physiol. Soc., Amer. J. Physiol.*, 1938, 123, p. 74.
3. GRINDLEY, G. C. Experiments on the influence of the amount of reward on learning in young chickens. *Brit. J. Psychol.*, 1929-30, 20, 173-180.
4. WOLFE, J. B., and KAPLAN, M. D. Effect of amount of reward and consummative activity on learning in chickens. *J. Comp. Psychol.*, 1941, 31, 353-361.

CHAPTER X

Habit Strength and the Time Interval Separating Reaction from Reinforcement

We have seen in the last two chapters that habit strength is dependent upon two measurable aspects of reinforcement: (1) the number of reinforcements and (2) the intensity (amount and quality) of the reinforcing agent. In the present chapter we shall consider the functional relationship of habit strength to a third measurable aspect of reinforcement—that of the time interval separating the reaction being conditioned from the reinforcing state of affairs (p. 80).

ORIGIN AND FRACTIONATION OF THE PROBLEM

The notion that the habit strength resulting from the conjunction of a receptor and an effector process is a function of the temporal nearness of a reinforcing state of affairs has long been current. It seems first to have been formulated by E. L. Thorndike in 1913, on the basis of general observation as an aspect of his "law of effect" (11, p. 173). Substantially the same idea was put forward, apparently independently, by Margaret Washburn in 1926 (14, p. 335). Thorndike expresses no opinion concerning the logical status of the principle. Washburn, however, clearly regarded it as a secondary, rather than a primary, principle. She believed it could be derived from the associative "law of recency," a supposed basic principle of learning once much in vogue but, in the light of recent work, now regarded primarily as a function of perseverative stimulus traces (p. 71).

Thorndike's original hypothesis breaks up into a number of distinguishable aspects which present convenient points of departure for a systematic consideration of the subject:

1. Is there, in fact, a functional dependence of habit strength upon the time interval separating the receptor-effector conjunction ${}_sC_r$ from the reinforcing state of affairs?

2. Assuming such a functional dependence, what is the direction of slope of the gradient?

3. How far does this gradient extend from the point of reinforcement before falling approximately to zero?

4. What is the shape of this gradient; i.e., what are its mathematical characteristics?

5. What parameters of the curve of learning are affected by the gradient of reinforcement—the rate of rise (F), or the limit of rise (m), or both?

6. Is the relationship a primary principle or is it a secondary one; i.e., can it be derived from other and more basic principles?

7. What other behavior processes, if any, are derivable as secondary phenomena from this principle?

EARLY DIRECT EXPERIMENTAL ATTACKS ON THE PROBLEM

A great deal of experimental effort has been devoted to the solution of one or another of the subsidiary problems growing out of Thorndike's original hypothesis. The first of these studies, published in 1917, was by John B. Watson (15). The apparatus of this experiment consisted essentially of a food chamber surrounded by sawdust to a depth of four inches. The task of the subjects, twelve hungry albino rats, was to dig through this sawdust, find a round hole giving access to the food chamber, and secure food which was in a shallow cup covered by a lid. Perforations in the lid allowed free passage of food odors. As preliminary training, the animals lived for a time in the food chamber, where they apparently ate freely from the food cup.

When the digging tests began the rats were divided into two groups, the animals of one group being allowed to eat as soon as they reached the food cup; but when the animals of the second group reached the food cup the perforated cover was held in place for 30 seconds before eating was permitted. In the course of 27 trials both groups of animals gradually reduced the time of reaching the food cup from around 100 seconds to six or seven seconds, though there were no indications of any special advantage in rate of learning of either group over the other.

The next experiment to throw much light on the problem was reported in 1929 by Mrs. Hamilton (née Haas) (5). She employed albino rats in a Warden compound Y-maze involving five successive choices, each of one correct or one incorrect turn. Between the last choice point of the maze and the food box a retention chamber was placed where the animals could be held as long as desired before being permitted to enter the food box and eat. Five

groups of approximately 20 animals each were used, the delays in the retention chamber employed with the respective groups being 0, 1, 3, 5, and 7 minutes.

A clearly marked difference in learning rate was found between the group permitted to eat at once and the various delay groups, but there was little indication of a consistent advantage in the shorter delay groups over those subjected to longer delays. All of the animals learned, the several delay groups requiring roughly

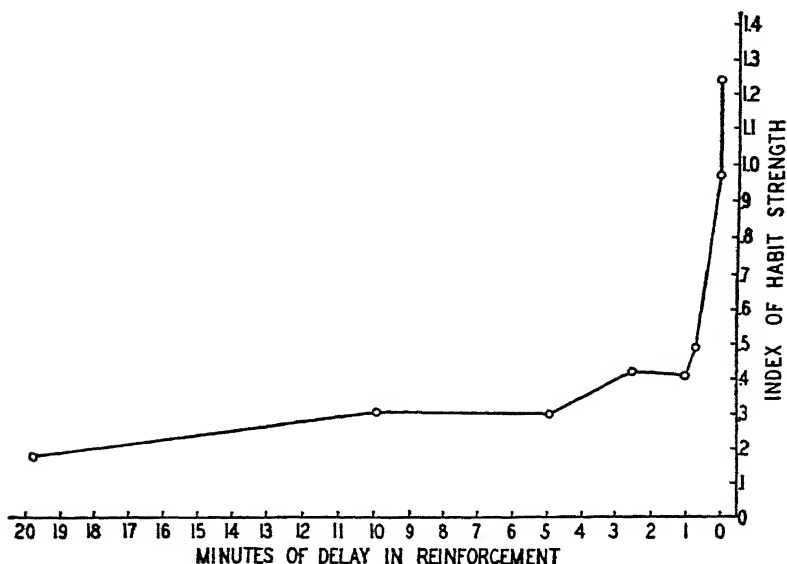


FIG. 30. Empirical delay-of-reinforcement gradient. The circles represent amount of learning for constant numbers of reinforcements as a function of the delay in the occurrence of the reinforcement. These values have been calculated from data published by Wolfe (16).

twice as many trials to reach a given criterion of learning as did the no-delay group. The Hamilton study, in contrast to that of Watson and an intervening experiment by Warden and Haas (13), accordingly indicates that (1) there is in fact a gradient of reinforcement, (2) the gradient slopes downward from immediate reinforcement as the length of delay increases (both quite as Thorndike and Washburn supposed), and (3) there is a suggestion that the gradient changes its nature in some important sense when delays of reinforcement exceed one minute. There is also an indica-

tion that the employment of a separate chamber for the restraint of the animal before it enters the food box is somehow critical in bringing out the influence of the period of delay on learning rate.

The next experimental investigation of the problem which demands our consideration was performed by Wolfe (16). The more significant portion of the Wolfe study concerns the learning of a single unit of a simple T-maze by eight groups, each of eight albino rats, with the following food delays for the respective groups:

0" 5" 30" 1' 2.5' 5' 10' 20'.

Wolfe employed special delay chambers, one for the final correct choice and one for the final incorrect choice, both being distinct

from the food chambers. Indices of habit strength calculated from Wolfe's published tables yield the values shown graphically in Figure 30. There it may be seen that the gradient falls sharply from delays of zero to those of one minute, after which the slope, though upon the whole continuous, is much more gradual. This study is in general agreement with Hamilton's findings in showing a critical change at a delay of around one minute; it also fills in the important gap lying between zero and 60 seconds by supplying evidence of the rate of learning at delays in reinforcement of five and 30

50 40 30 20 10
SECONDS OF DELAY IN REINFORCEMENT (t)

FIG. 31. The first four of the Wolfe delay-of-reinforcement values, together with the negative growth or decay function which fits them rather well. This curve represents a decrease of between 1/15 and 1/16 at each additional second of delay in reinforcement.

seconds respectively. The values in this region when carefully examined turn out to approximate very closely a negative growth function, as shown in Figure 31. This gives us the first convincing clue concerning the answer to our third and fourth questions formulated above.

PERIN'S EXPERIMENT

The most recent experiment in this field is reported by Perin (10). In that portion of his investigation which especially concerns us here, Perin employed a modified form of the Skinner box (see p. 87 above). Through a horizontal slot in a metal plate on one wall of the experimental chamber there projected an easily

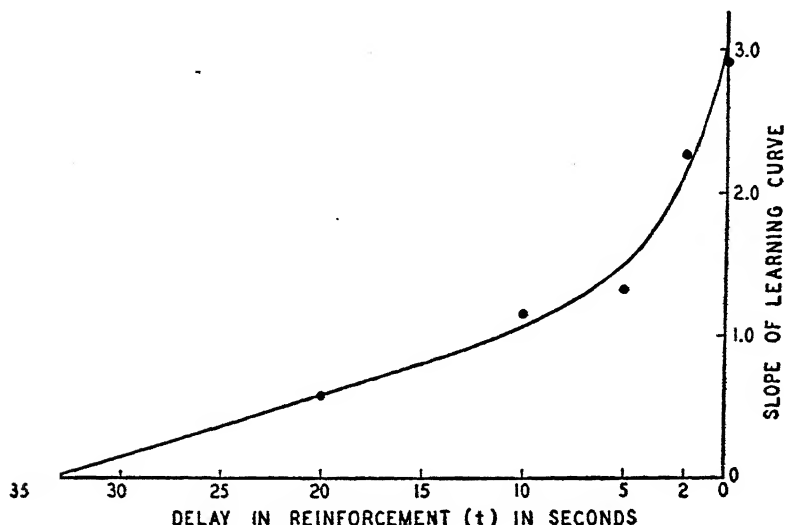


FIG. 32. Graphic representation of the gradient of reinforcement as indicated by the slopes (tangents) of the composite learning curves of five of Perin's groups of animals at the point where 50 per cent of the trials were without "error." The slope of each group is represented by a circle; the curved line running through the circles is a special form of negative growth function which was fitted to these values. (Reproduced from Perin, 10.)

moved brass rod. During the habituation period the apparatus was so set that a movement of this rod a few millimeters to either the right or the left would immediately deliver a pellet of food to the food cup beneath. After the animal had learned to perform both acts with some facility and his preference for one of them had been determined, the setting of the apparatus was so changed that (1) a movement of the rod in the preferred direction gave *no* food, and (2) a movement of the rod in the non-preferred direction caused (a) the rod instantly and silently to be withdrawn and

(b) the delivery of a pellet of food after time intervals varying according to the group involved, as follows:

0" 2" 5" 10" 20" 30".

As a rule the animals sat quietly by the food cup after the withdrawal of the bar, and simply waited for the food to be delivered. It is not without significance that many of the animals of the 30-second group ceased to operate the bar after varying numbers of trials; because of this the results of these animals could not be employed in the plotting of Perin's gradient of reinforcement.

The results of this experiment which are of special interest in the present context are shown graphically in Figure 32. There it may be seen that the rates of learning, as indicated by the slopes of the learning curves of the several groups of animals at the 50 per cent level of correctness, show a negatively accelerated descending gradient much as does Wolfe's study (Figure 31). There is, however, this difference: the slope of Perin's curve is of such a nature that, when extrapolated, it falls to zero at 34 seconds; with a somewhat different method of plotting the learning curve from which the tangents were taken, the extrapolated gradient falls to zero at 44 seconds.

THE RECONCILIATION OF SOME EXPERIMENTAL PARADOXES

The most outstanding paradox encountered among the experimental results outlined above is the fact that the Watson study and a comparable one by Warden and Haas (13) show no gradient of reinforcement, whereas the Hamilton investigation and, particularly, that of Wolfe clearly indicate such a gradient. As already suggested, the difference in the outcomes of the two groups of studies is probably to be attributed mainly to the fact that in the first two the retention or delay occurred in the food chamber, whereas in the latter two it took place in a separate (non-feeding) compartment.

Actually, on the basis of secondary reinforcement, we should expect exactly such a difference in the outcome of the two groups of experiments. It may be recalled (Chapter VII, p. 97) that any stimulus which is closely and consistently associated with a reinforcing state of affairs will itself gradually acquire the power of secondary reinforcement regardless of whether the transmitting stimulus is primarily or secondarily reinforcing. Thus in the Wat-

son experiment and also in the Warden and Haas study the food chamber would largely have acquired this power from the preliminary or habituation training for both groups of animals alike. By the same principle, the odor of the food which came through the perforations in the food-cup lid in both experiments had already acquired secondary reinforcing power as the result of the animals' having eaten the food, even before habituation to the food chamber. There was therefore, on two counts, no delay in the effective (secondary) reinforcement in either group and so, naturally, it is not to be expected that a delay of 30 seconds in the actual eating would produce a retardation in the learning rate large enough to be detected by the use of small groups of animals.

Turning, now, to the Hamilton and Wolfe studies we still find, even with the separate retention chamber, the conditions which are both necessary and sufficient for secondary reinforcement, though in an attenuated form. In these investigations the eating of the food is immediately associated with the food cup and the food chamber by both groups alike, so that all parts of the food chamber as stimuli must gradually acquire secondary reinforcing powers as the trials increase in number. Next, since the stimuli arising from the door giving access to the food chamber are associated closely with the food chamber itself, this door must gradually acquire secondary reinforcing powers following the acquisition of these powers by the food chamber as a whole. In a similar manner, the entire retention chamber would gradually acquire a measure of secondary reinforcing power from reinforced association with this door, though presumably the longer the delay there, together with the incidental irrelevant activity during the periods of delay, the slower would be this process. This probably explains the fact that Hamilton's animals continued to learn with fair speed under delays of reinforcement up to seven minutes, and Wolfe's animals, under delays up to twenty minutes.

Moreover, considering the simplicity of Wolfe's maze, which involved only a single choice, his animals learned notably more slowly than did Hamilton's, which were required to learn five choices. This paradox quite probably was due in part to the fact that Wolfe employed a retention chamber on the false choices as well as on the correct ones. Presumably the two retention chambers were physically identical. Under these conditions the secondary reinforcing power acquired by the retention chamber next to the food box would generalize (p. 183) to the one outside the non-

reward box, and so tend at first to reinforce the false choice and thereby retard the learning. In addition, the extinction effects arising from the non-reinforcement of the incorrect final choices would generalize (p. 264) to the correct choice to some extent, thereby weakening that reaction and still further retarding the learning. Also presumably contributing to the relative slowness of the learning of Wolfe's animals is the fact that inside the retention chamber on the non-reward arm of the maze was a dish containing food from which the animal was excluded by a wire-screen cover. In all probability the odors and other associated stimuli arising from this non-eating food situation constituted powerful secondary reinforcing influences, as in the Grindley experiment (p. 126), which also would tend to reinforce the false choice until extinction should supervene.

Finally, there is reason to believe that in all of the above studies, but especially in the Wolfe study, there enters the complicating factor of spatial orientation. This is the capacity possessed by most organisms to return to a point of reinforcement if the distance is not too great. The mechanisms mediating this behavior are complex and cannot be taken up in this place.

The conditions of Perin's experiment, on the other hand, were designed in such a way as to preclude all irrelevant secondary reinforcement as well as all irrelevant spatial orientation. This presumably accounts for the fact that (1) the extrapolation of Perin's gradient falls to zero, whereas the extrapolation of Wolfe's gradient lacked much of doing this; and (2) Perin's gradient reaches zero at a delay of between 30 and 40 seconds, whereas Wolfe's gradient continues to fall for 20 minutes, and the asymptote of this fall lacks much of being zero.

FORMULATION OF THE GOAL GRADIENT HYPOTHESIS

As we have just seen, Perin's investigation suggests that, uncomplicated by irrelevant factors, the basic temporal gradient of habit strength as a function of the delay in reinforcement in the case of the albino rat actually extends over a relatively short period of time, possibly no more than 30 seconds and very probably less than 60 seconds. On the other hand, the Wolfe study indicates that under ordinary learning conditions, where plenty of opportunity for secondary reinforcement usually exists, the gradient may extend in considerable force for a relatively long period. This means that

what was originally regarded as a single principle has turned out upon intensive investigation to involve two fairly distinct principles: (1) the short gradient reported by Perin, which will be called the *gradient of reinforcement*, an expression coined by Miller and Miles (9); and (2) the more extended gradient which is presumably generated as a secondary phenomenon from Perin's gradient of reinforcement acting in conjunction with the principle of secondary reinforcement. This second and more extended gradient may with some propriety retain the original name of the *goal gradient*, an expression employed by the present author in his first discussion of the subject (6).

Unfortunately it is not yet clear in exactly what quantitative manner the basic gradient of reinforcement, in combination with secondary reinforcement, generates the more extended goal gradient. We do, however, have a number of promising leads. Because of the intimate relation known to exist between the conditioning of a stimulus to a reaction and the acquisition by that stimulus of secondary reinforcing power (p. 100), it is plausible to assume that stimuli acquire this power according to the primitive gradient of reinforcement demonstrated by Perin. It is also assumed that once a stimulus has acquired a certain amount of the capacity for secondary reinforcement, this will immediately begin to operate according to Perin's primitive gradient of reinforcement to reinforce antecedent receptor-effector connections and to endow each newly associated receptor process with secondary reinforcing powers, and so on. Thus the goal gradient would result from the summation of an exceedingly complex series of overlapping gradients of reinforcement, in part consisting of, but largely derived from, the "primary" reinforcement occurring at the end of the temporal period covering the behavior sequence involved. Also, the generation of overlapping secondary gradients presumably would take place under ordinary learning conditions not only beyond the range of Perin's primitive gradient of reinforcement, but within its range as well, so that the period preceding "primary" reinforcement by 30 or 40 seconds would present a picture not very different from other portions of the total range, though Wolfe's results (Figures 30 and 31) suggest that the characteristics of the goal gradient for the first minute of the delay in reinforcement may differ somewhat from the more remote portions. In view of the all but universal prevalence of the conditions which generate secondary reinforcement, coupled with the great difficulty experienced by Perin in

eliminating them from his experiment, it is fairly evident that the principle immediately concerned in ordinary learning situations is what we have called the goal gradient, whereas both the gradient

TABLE 2

THIS TABLE SHOWS THE THEORETICAL HABIT STRENGTHS IN HABS OF RECEPTOR-EFFECTOR CONJUNCTIONS WITH UNLIMITED PRACTICE WHEN THE REACTION IS FOLLOWED BY REINFORCEMENT AFTER VARYING AMOUNTS OF DELAY IN SECONDS (t). IT IS ASSUMED THAT WITH ZERO DELAY THE REINFORCING AGENT EMPLOYED WOULD YIELD A HABIT STRENGTH (M') OF 80 HABS AT THE LIMIT OF PRACTICE, AND THAT EACH ADDITIONAL SECOND OF DELAY REDUCES THE LIMIT OF HABIT STRENGTH BY 1/65TH.

Amount of Delay (t)	Habit-Strength Limit (m')	Amount of Delay (t)	Habit-Strength Limit (m')	Amount of Delay (t)	Habit-Strength Limit (m')
0	80.00	30	50.29	60	31.61
1	78.77	31	49.52	65	29.26
2	77.56	32	48.76	70	27.08
3	76.37	33	48.01	75	25.07
4	75.20	34	47.27	80	23.20
5	74.04	35	46.55	85	21.48
6	72.91	36	45.83	90	19.91
7	71.79	37	45.13	95	18.39
8	70.68	38	44.44	100	17.02
9	69.60	39	43.75	105	15.76
10	68.53	40	43.08	110	14.58
11	67.48	41	42.42	115	13.50
12	66.44	42	41.77	120	12.49
13	65.42	43	41.13	125	11.56
14	64.42	44	40.50	130	10.70
15	63.43	45	39.87	140	9.17
16	62.46	46	39.26	150	7.85
17	61.50	47	38.66	160	6.73
18	60.45	48	38.07	170	5.76
19	59.62	49	37.48	180	4.94
20	58.71	50	36.90	190	4.23
21	57.81	51	36.34	200	3.62
22	56.92	52	35.78	210	3.10
23	56.04	53	35.23	220	2.66
24	55.18	54	34.69	230	2.28
25	54.34	55	34.16	240	1.95
26	53.50	56	33.63	270	1.23
27	52.68	57	33.12	300	.77
28	51.87	58	32.61	330	.49
29	51.07	59	32.11	360	.31

of reinforcement and secondary reinforcement are represented in the goal gradient, which they presumably generate.

In the interest of immediate utilization we accordingly proceed to the consideration of the more detailed characteristics of the goal

gradient. On the basis of the direct experimental approaches outlined above, together with certain indirect approaches presently to be disclosed, we formulate our hypothesis concerning the molar functional relationship of habit strength to the temporal delay in reinforcement as follows: (1) *The maximum habit strength (m') attainable with a given amount and quality of reinforcement closely approximates a negative growth function of the time (t) separating the reaction from the reinforcing state of affairs;* (2) *the asymp-*

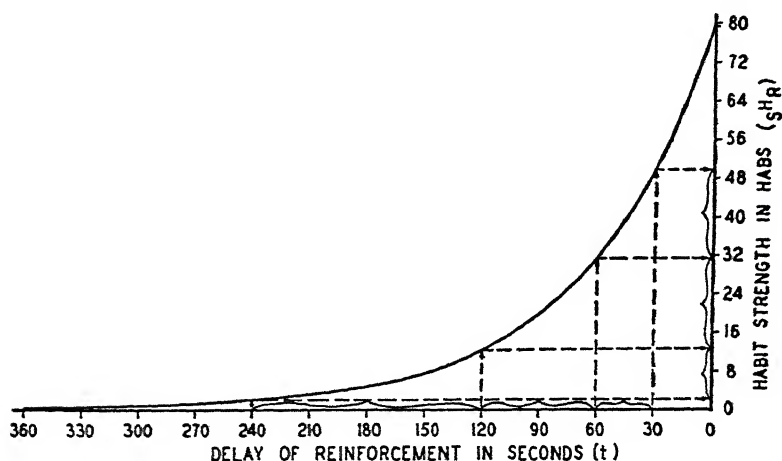


FIG. 33. Theoretical goal gradient plotted from the values shown in Table 2. This curve is drawn on the assumption that the value of habit strength at the limit of practice is reduced 1/65th for each additional second of delay in reinforcement. The braces on the vertical scale show the theoretical difference in habit strength which would be produced by each at the limit of practice. Note (1) that none of these differences is like any of the others, and (2) that the middle difference is the greatest of the three.

tote or limit of fall of this gradient is zero; and (3) the more favorable the condition for the action of secondary reinforcement, the slower will be the rate of fall, so that this limit may not be approximated until after considerable periods of delay, though for many conditions less favorable for secondary reinforcement it may be reached in a period of from 30 to 60 seconds.

For purposes of precise illustration, the values of such a negative growth function have been calculated and are reproduced as Table 2, on the assumption that the conditions of secondary reinforcement are such as to bring the gradient practically to zero at a delay in "primary" reinforcement of about six minutes. The

values appearing in this table are represented graphically by the curve shown in Figure 33.

With a definite hypothesis as to the quantitative characteristics of the goal gradient available, the possibility at once arises of deriving from it, and the other principles of the system, numerous implications in the form of corollaries. A comparison of these deductions with relevant experimental evidence then affords a basis for the acceptance, rejection, or further modification of the hypothesis. With good fortune, this indirect procedure, supplementing that of the direct experimental attack already considered, may be expected to lead to the isolation of a sound and comprehensive scientific principle more quickly than would either approach employed alone.

ORGANISMS GRADUALLY ACQUIRE A PREFERENCE FOR THE ACT FOLLOWED BY THE SHORTER DELAY IN REINFORCEMENT

Let it be assumed that hungry albino rats are presented with a choice of two short passageways; at the end of each is an exactly similar food reinforcement. In both alleys alike there is, next to the food box, a delay chamber in which the animal is retained for a certain period before being permitted access to the food. More specifically, let it be assumed that the delay in one chamber is 30 seconds and that the delay in the other is 60 seconds. Finally, during training one or the other alley is always blocked, the order of the reinforcements on the respective alleys being randomized in such a way as to keep approximately the same number of reinforcements on each at all times. Therefore, at any point in the training at which it is desired to know the relative strengths of the two habits thus set up, the entrance to both alleys may be left open, at which time a competition between the two habit strengths will occur, the stronger habit of course dominating. By training groups of comparable animals with different numbers of reinforcements before the testing, it would be possible to determine relatively how strong the two habits are at various stages of training, and in this indirect manner verify empirically the goal gradient hypothesis formulated above.

Table 2 shows that the theoretical limit (m') of habit strength at a delay of 30 seconds is 50.29 habs, and the limit at 60 seconds is 31.61 habs. With these asymptotes of the positive growth, or learning, curves available, and taking our fractional increment (F)

TABLE 3

COLUMNS 2, 3, AND 4 OF THIS TABLE SHOW THE THEORETICAL HABIT STRENGTH AT THE SUCCESSIVE REINFORCEMENTS BY THE SAME REINFORCING AGENT WHERE THE FRACTIONAL INCREMENT (F) IS 1/20, AND WHERE THE DELAY IN REINFORCEMENT IS 30", 60", AND 90" RESPECTIVELY. THE m' VALUES EMPLOYED IN THE COMPUTATION OF THESE sH_R VALUES WERE TAKEN FROM TABLE 2. THE VALUES IN COLUMNS 5 AND 6 REPRESENT THE PER CENT OF TEST TRIALS IN WHICH THE HABIT WITH THE SHORTER DELAY IN REINFORCEMENT WOULD BE EXPECTED TO DOMINATE ON THE ASSUMPTION THAT THE RANGE OF OSCILLATION HAS A STANDARD DEVIATION OF 13 HABS.

Ordinal Number of Reinforcement	Strength of sH_R in Habs at 30" Delay in Reinforcement	Strength of sH_R in Habs at 60" Delay in Reinforcement	Strength of sH_R in Habs at 90" Delay in Reinforcement	Per Cent of Trials at Which the 30" Habit Dominates Over 60"	Per Cent of Trials at Which the 30" Habit Dominates Over 90"
1	2	3	4	5	6
0	0.0	0.0	0.0	50.0	50.0
1	2.5	1.6	1.0	52.0	53.3
2	4.9	3.1	1.9	53.9	56.4
3	7.2	4.5	2.8	55.8	59.3
4	9.3	5.9	3.7	57.5	62.0
5	11.4	7.2	4.5	59.1	64.6
6	13.3	8.4	5.3	60.6	66.9
8	16.9	10.6	6.7	63.4	71.1
10	20.2	12.7	8.0	65.8	74.6
12	23.1	14.5	9.2	68.0	77.6
15	27.0	17.0	10.7	70.7	81.2
18	30.3	19.1	12.0	73.0	84.0
21	33.2	20.8	13.1	74.9	86.2
25	36.3	22.8	14.4	76.9	88.4
30	39.5	24.8	15.6	78.8	90.3
35	41.9	26.4	16.6	80.2	91.6
40	43.8	27.5	17.4	81.2	92.5

at 1/20, there may be generated the progressive learning values shown in columns 2 and 3 of Table 3. For purposes of ready comparison, these learning curves are represented graphically in Figure 34. It will be noted at a glance that the 30-second curve gradually rises above the 60-second curve, the distance separating them increasing as the number of reinforcements increase. This yields our first corollary:

1. *The shorter the delay in reinforcement, the steeper becomes the rise of the associated curve of learning.*

To be of critical scientific value, a theoretical deduction should lead to the possibility of comparison with a relevant empirical

observation. Unfortunately, Corollary I as it stands does not permit an observational check because habit strength, as such, cannot be observed. However, by combining it with the well-known principle that the greater the habit strength, the shorter will be the time of reaction evocation (p. 105), we easily derive a second corollary which is susceptible of such verification:

II. *When a reaction is reinforced after a short delay, the time required to execute the act will be less than that required to execute*

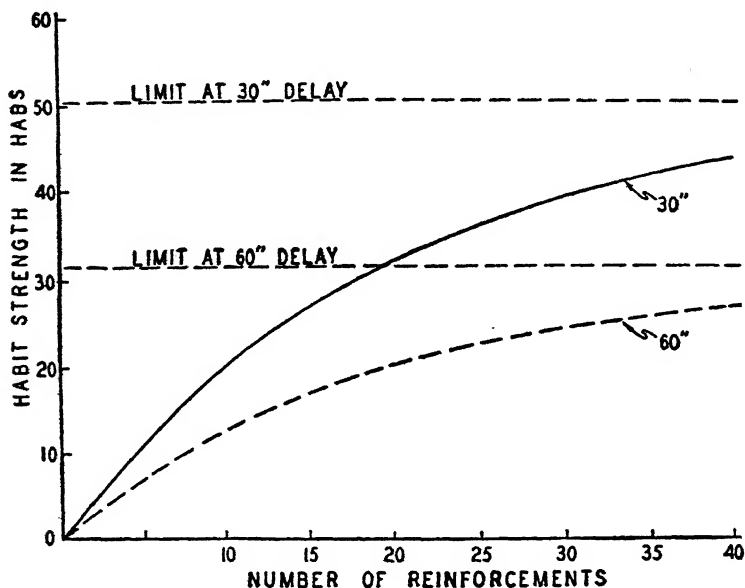


FIG. 34. Parallel theoretical learning curves with the same rate of rise ($F = 1/20$) but with different asymptotes as determined by the respective periods of delay in reinforcement (30 and 60 seconds) as shown in Table 2. The above curves were plotted from columns 2 and 3 of Table 3.

a comparable act which has had the same number of reinforcements but in which the delay of the reinforcements has been longer.

Empirical confirmation of the essential soundness of Corollary 2 is furnished by a number of studies, notably one reported by Anderson (2). This experiment was set up (1) in substantially the manner postulated in the theoretical arrangement just described, with the exception that the animals were permitted to choose freely between the two entrances throughout the training process; the investigator's labor was thereby greatly economized. Early in the

training this procedure would, of course, begin to give a disproportionately large number of reinforcements to the reaction associated with the shorter delay. While this doubtless weakened, relatively, the act involving the longer delay in reinforcement, it probably did not materially change the outcome so far as the present set of corollaries is concerned.

Anderson's animals had to cross a platform on the way to the retention chambers, the distance amounting to seven feet. It happens that in this study two pairs of delays are reported which are alike in their ratio (1:3), and closely similar in their empirical discriminability (83 per cent and 80 per cent after 40 reinforcements), yet the lengths of the delays are very different: 10 and 30 seconds as compared with 120 and 360 seconds, the lengths of the second pair of delays being twelve times those of the first. The mean runway times for each of the eight days of training of the respective groups of

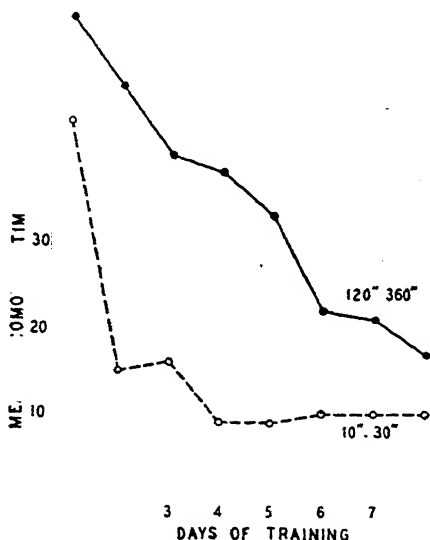
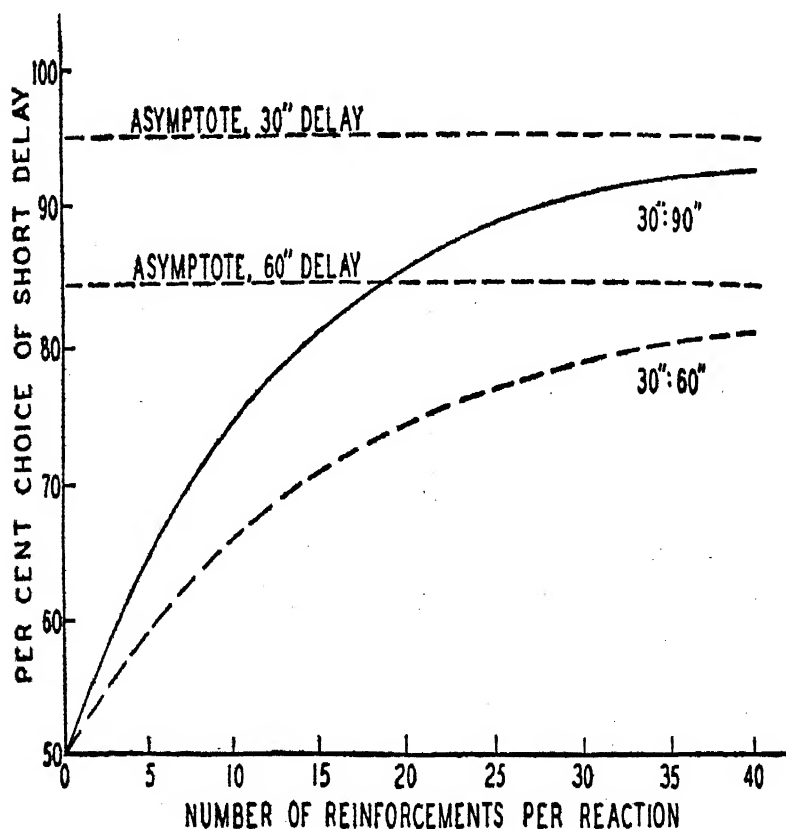


FIG. 35. Parallel curves showing differing locomotor times for the same distance, training, and discriminability, but markedly different delay in reinforcement. (Plotted from Anderson's published Table 1, 2, p. 424.)

animals are shown in Figure 35, where it may be seen at a glance that the acts associated with the pair of short delays have a lower mean reaction time throughout the entire training period, which thus agrees with the corollary.

At this point we must introduce a factor to be taken up in detail a little later—that of the spontaneous oscillation or variability in habit strength (p. 304 ff.). It will be sufficient here to say only that there is reason to believe that the effective strength of all habits when functioning as reaction potentials is subject to continuous uncorrelated interferences, presumably mainly from processes arising spontaneously within the nervous system, and that

the magnitudes of these disturbances are distributed approximately according to the "normal" law of probability. On the assumption that the oscillations in habit strength are largely uncorrelated, it follows that the weaker of two competing habit tendencies will frequently be depressed only slightly when the stronger one chances to be depressed relatively much, with the result that the weaker habit will dominate on that occasion. Indeed, this failure of the



There it will be seen that, theoretically, at first the two choices are equally likely, i.e., the entrance to the alley leading to the chamber associated with the 30-second delay should occur on only 50 per cent of the test trials because the two probability distributions coincide exactly. However, as practice progresses the choice associated with the shorter delay gradually attains an advantage

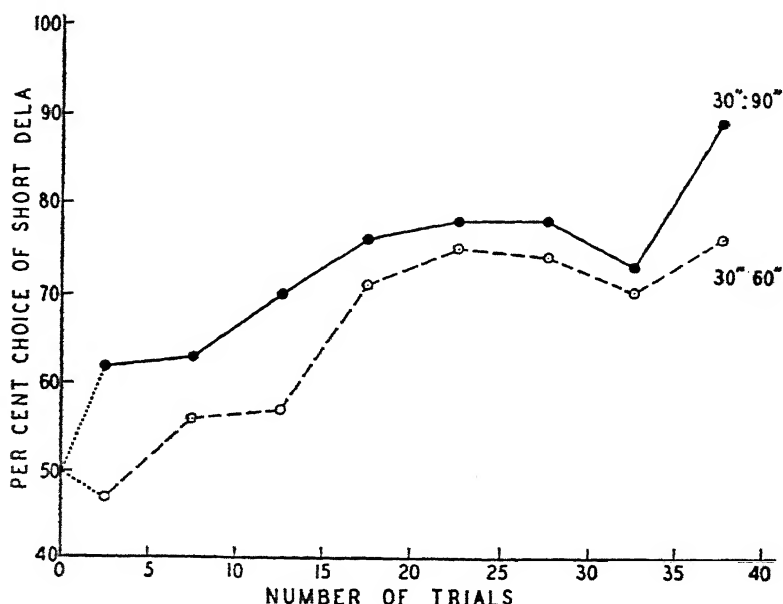


FIG. 37. Graph showing empirical curves of increasing preferences for the reaction involving the shorter of two delays in reinforcement. (Plotted from tables published by Anderson, 1.)

which, after 40 pairs of reinforcements, reaches the considerable amount of 81.2 per cent.

On the basis of the above calculations we generalize and formulate the following corollaries:

III. *With training, organisms tend to choose that one of a pair of alternative acts which yields reinforcement with the lesser delay.*

IV. *The preference for that one of a pair of acts involving the lesser delay in reinforcement is attained gradually as training increases.*

Empirical confirmation of Corollaries III and IV is seen in the study reported by Anderson (1). This investigator found that in

the course of 40 trials involving delays of 30 and 60 seconds respectively, eight albino rats displayed a gain in the choice of the act involving the 30-second delay which extended from 47 per cent (approximately chance) to 76 per cent. The curve of this learning is shown in the lower graph of Figure 37. Moreover, DeCamp (3), Yoshioka (17), and Grice (4) have all found substantially the same relationship to hold where the delay in reinforcement was incidental to the difference in length of two alternative paths leading to a reinforcement. It is noteworthy that Grice's experiment was set up in such a way as to keep the number of reinforcements on the two paths more nearly equal than was the case in any of the other studies so far reported; his results are therefore more comparable to the conditions presupposed by the above deductions.

THE RATE OF DISCRIMINATION OF ACTS AS A FUNCTION (1) OF
THE DIFFERENCE IN THE DELAYS INVOLVED AND (2) OF
THE ABSOLUTE MAGNITUDE OF THE DELAYS

Our next problem concerns the relative rate of preference acquisition of alternative reactions involving differential delays in reinforcement as a function of the amount of difference in the two delays. Let us take, for example, the same theoretical arrangement assumed above, with the exception that the delays are of 30 and 90 seconds respectively, which gives the organism the relatively coarse ratio of 1 to 3 instead of 1 to 2, as previously. The theoretical course of the acquisition of habit strength with a 30-second delay in reinforcement may be seen in the second column of Table 3, and that of a 90-second delay, in the fourth column. The per cent of the trials in which the act associated with the 30-second delay of reinforcement would be expected to dominate over that associated with the 90-second delay is given in the sixth column and is represented graphically in the upper (solid) curve of Figure 36. There it may be seen at a glance that the larger difference yields the more rapid acquisition of dominance by the act involving the lesser delay of reinforcement. Specifically, at the fifteenth reinforcement the discrimination involving the 30-90-second delay reached the level of dominance attained at the fortieth reinforcement by the one involving the 30-60-second delay. Generalizing, we arrive at our fifth and sixth corollaries:

V. Other things equal, the greater the difference in the delay of reinforcement of two competing reactions, the less will be the train-

ing required to give the act involving the lesser delay a given degree of dominance.

VI. *Other things equal, the coarser the ratio of the delay of reinforcement of two competing reactions, the less will be the training required to give the act involving the lesser delay a given degree of dominance.*

Corollaries V and VI also find ready empirical verification in the investigation reported by Anderson (1). For purposes of easy comparison, the experimental learning scores of Anderson's 30-60-second group of animals (a ratio of 1 to 2) are presented graphically in Figure 37, in parallel with the results from his 30-90-second group (a ratio of 1 to 3). While somewhat irregular, as is to be expected from the relatively small number of animals employed in the respective groups, the general relationship shown by the two theoretical curves of Figure 36 is discernible. The 30-90-second discrimination reached, between 15 and 20 reinforcements, a degree of short-delay dominance only attained by the 30-60-second combination between the 35th and 40th reinforcements. Completely parallel results were obtained by Yoshioka (17) in the discrimination of pairs of alternate paths to a goal. He reports that a given

TABLE 4

TABLE SHOWING THE PER CENT OF CHOICES OF ACTS ASSOCIATED WITH THE SHORTER OF TWO DELAYS TO BE EXPECTED ON THEORETICAL GROUNDS AND THE PARALLEL EMPIRICAL VALUES REPORTED BY ANDERSON (1, p. 54).

Ratio	Delays of Reinforcement Compared	Theoretical Per Cent Choices of Act Involving Shorter Delay	Empirical Per Cent Choices of Act Involving Shorter Delay
1 : 3	120":360"	71.8	82
1 : 3	60":180"	89.7	87*
1 : 3	30":90"	92.5	89
1 : 3	10":30"	80.6	80
1 : 2	120":240"	69.1	70
1 : 2	60":120"	81.8	85
1 : 2	30":60"	81.2	76
1 : 2	10":20"	67.9	74
1 : 1.5	120":180"	64.0	63
1 : 1.5	60":90"	71.0	66
1 : 1.5	30":45"	68.9	...
1 : 1.5	10":15"	59.6	...

* At one point in Anderson's article this value is given as 84 per cent, but in the original table, as well as in his thesis on file in Yale University, the mean of which has been checked, the value is 87 per cent.

amount of training on two alternative alleys 210 and 233 inches in length (a difference of 23 inches and a ratio of about 1 to 1.11) yielded a preference for the shorter path of 10.80 units, whereas when the 210-inch alley was paired with a 276-inch alley (a difference of 66 inches and a ratio of about 1 to 1.3), the same amount of training yielded a preference for the shorter alley of 18.85 units.

Our next problem concerns the relative ease of discriminating acts involving a given amount of difference in the delay of reinforcement as dependent upon whether the length of the shorter of each pair of delays is absolutely short or long. For example, what does our hypothesis imply as to the ease of differentiating acts associated with delays of 30 and 60 seconds as compared with those of 60 and 90 seconds when both pairs of delays involve exactly the same absolute difference, 30 seconds? We have already seen (Table 3 and Figure 36) that theoretically 30 and 60 seconds yield at 40 reinforcements 81.2 per cent choice of the 30-second act. Table 4 shows that at 40 reinforcements the 60-second choice over the 90-second choice, theoretically, occurs on 71.0 per cent of the trials. But,

$$81.2 > 71.0.$$

Generalizing, we arrive at our seventh corollary:

VII. *Equal differences in the delays of reinforcement of two competing acts lead with equal practice to a lower per cent of preferential choices of the act associated with the shorter delay when the two delays are large (i.e., when the ratio is relatively coarse) than when they are small (i.e., when the ratio is finer).*

This corollary also finds complete empirical confirmation. In the Anderson study already cited, it was found (column 4, Table 3) that at 40 reinforcements delays of 30 and 60 seconds gave 76 per cent preference for the 30-second act, whereas delays of 60 and 90 seconds gave a preference of only 66 per cent to the 60-second act. Similarly, Grice found that alternative alleys of six feet and twelve feet, with a six-foot difference, were discriminated in a mean of 12.4 trials, whereas alleys of 24 feet and 30 feet with exactly the same difference were discriminated only after a mean of 28.4 trials, i.e., after about twice as much training.

THE RELATION OF THE DELAY IN REINFORCEMENT TO WEBER'S LAW

Experimental results such as those just summarized under Corollaries VI and VII have led to the view that the discrimination

between intervals of delay in reinforcement as found by Anderson (1) and between alternative paths to reinforcement as found by Yoshioka (17) indicates the conformity of these learning processes to Weber's law. Indeed, influenced largely by the views of Yoshioka, the present writer first postulated the gradient of reinforcement as being a logarithmic function of the delay of reinforcement, which is implicit in the Weber's law hypothesis; further consideration, which revealed certain mathematical paradoxes arising from the nature of the logarithmic function (7, p. 273) led to its abandonment in favor of the exponential or negative growth function represented in Table 2 and Figure 33.

This problem brings us to our eighth corollary. Weber's law, as applied to the delay in reinforcement, requires that all pairs of delays of equal ratios, e.g., 1 to 2, with equal amounts of training, yield equal per cents of preference for the act associated with the shorter delay. The present (exponential) hypothesis leads to quite different expectations. Suppose that we have four pairs of acts, all with delays in the ratio of 1 to 2 as follows:

10'' vs. 20''; 30'' vs. 60''; 60'' vs. 120''; 120'' vs. 240''.

By Table 2, these pairs of delays *at the limit of practice* generate the following habit strengths:

68.53:58.71; 50.29:31.61; 31.61:12.49; 12.49:1.95.

Appropriate computations based on the same assumptions as outlined above show that at 40 reinforcements the respective situations would generate the following pairs of habit strengths:

59.71:51.15; 43.81:27.54; 27.54:10.88; 10.88:1.70.

Further calculations show that these pairs of habit strengths correspond to the following per cents of preference for the shorter delays:

10'':20'' = 67.9 per cent
 30'':60'' = 81.2 per cent
 60'':120'' = 81.8 per cent
 120'':240'' = 69.1 per cent

Comparable computations have been made for a series of delays which stand in a coarser ratio of 1 to 3 and in a finer ratio of 1 to 1.5. The theoretical outcome of all three sets of delays has been drawn up systematically in Table 4.

An examination of the third column of this table shows that according to the present set of hypotheses, it is not to be expected that all pairs of acts whose delays of reinforcement stand in the same ratio will be equally discriminable. On the contrary, it is evident that while the several entries under a given ratio show some resemblance, they also show marked differences. Moreover, these differences manifest a characteristic pattern. First, there is a central point of maximum discriminability from which the ease of discrimination decreases as the absolute magnitude of the delays either increases or decreases; thus in the ratio of 1:3 the combination of 30-90 seconds gives a maximum of 92.5 per cent, whereas that of 60-180 seconds, a pair of larger values, gives 89.7 per cent, and that of 10-30 seconds, a pair of smaller values, gives 80.6 per cent. Second, as the ratio of the delays grows finer, the point of maximum ease of discrimination shifts in the direction of the longer delays; thus the maximum ease of discrimination falls in the 1:3 ratio on the combination of 30-90 seconds, whereas at the 1:2 ratio it falls on the larger values of 60-120 seconds.

These *a priori* expectations find striking empirical confirmation in the experimental results reported by Anderson (1). The relevant empirical values have been arranged in column 4 of Table 4, in parallel with the theoretical values. There it may be seen that (1) the discriminability of acts associated with different delays in reinforcement is in fact *not* equal, i.e., Weber's law does not hold; (2) the combination 30-90 seconds has the maximum ease of discriminability of the 1:3 ratio exactly as calculated, from which the ease of discrimination falls off in both directions; (3) the combination 60-120 seconds has the maximum ease of discriminability of the 1:2 ratio, a shift in the direction of the larger values, from which the ease of discrimination diminishes in both directions, again exactly as demanded by the theory.

A similar outcome in favor of the exponential and opposed to the logarithmic relationship was obtained by Grice (4) in the ease of discriminating different ratios of alternate paths to food reinforcement. While Grice's experiment was not designed in such a way as to bring out the exponential relationship as dramatically as does Anderson's study, this characteristic in his results was effectively demonstrated by means of a process of curve fitting.

Generalizing from the preceding observations, we may formulate our eighth corollary as follows:

VIII. *The ease of discrimination of acts associated with different delays of reinforcement has for a given ratio of delay lengths a maximum occurring at a central point of absolute lengths, from which point it diminishes progressively with both increase and decrease in the absolute magnitude of the delays, the point of maximum ease of discrimination shifting in the direction of the longer delays as the ratio of the two delays grows finer.*

Extrapolating from the same general considerations, we may formulate a ninth corollary:

IX. *With the ratio between delays of reinforcement constant, discrimination becomes impossible when the periods of delay involved become sufficiently great or sufficiently small.*

SUMMARY

The favorite method employed by experimentalists in determining the functional relationship of the rate of learning to the delay in reinforcement has been to present an organism with the possibility of performing two alternative acts one of which receives reinforcement after a shorter delay than the other. The organism is then permitted gradually to develop a preference for the act involving the shorter delay by a process of trial and error, the number of trials required to reach a given preference criterion being taken as an indication of the ease of the "discrimination." In the purer forms of these experiments the acts are strictly comparable, e.g., merely turning to the right or the left and walking a few inches or feet. The alternative acts of some experiments, however, require the organism to traverse different lengths of paths to reach the point of reinforcement, in which case not only is there presumably involved the delay required to traverse a given path, but there is introduced a third, though closely parallel factor, the amount of work or energy expenditure required in traversing the different distances (see p. 280 ff.).

The early experimental work on the relation of the rate of learning to the extent of the delay in reinforcement yielded negative results. This was apparently because in the procedures employed, the food box served as the retention chamber, which introduced in a gross manner the factor of secondary reinforcement. When a separate compartment adjoining the food box was used as a retention chamber, the spread of secondary reinforcing tendencies was sufficiently gradual to show in one study, that of

Wolfe, a negatively accelerated falling gradient extending to delays of reinforcement 20 minutes in duration. Perin, on the other hand, in an experiment set up in such a way as to exclude secondary reinforcement as completely as possible, found a negatively accelerated gradient of rate of learning which fell to zero at a delay of only 30 seconds or so.

The most plausible interpretation of these superficially conflicting results seems to be (1) that the basic or primary *gradient of reinforcement* is only about 30 seconds in duration, and (2) that under ordinary learning conditions secondary reinforcement combines with the gradient of reinforcement to produce a derived phenomenon which may be called the *goal gradient*.

Partly on the basis of direct experimental evidence, but mainly on the grounds of indirect evidence, it is concluded that the most plausible hypothesis concerning the quantitative characteristics of the goal gradient is: In situations where both (1) the primary gradient of reinforcement and (2) the principle of secondary reinforcement are operative in a progressive manner, *the goal gradient is an exponential or negative growth function, and the greater the influence of secondary reinforcement, the less steep will be the slope of the gradient.*

From this postulate or hypothesis, together with other principles of the system, a number of corollaries follow. The first of these is that, other things equal, the shorter the delay in the reinforcement of a given act, the steeper will be the curve of learning of that act and so, at any given number of reinforcements, the shorter will be the time required to execute the act; this is in agreement with experimental findings.

In the typical trial-and-error situation involving alternative acts with different delays in the reinforcement of each, it follows from the goal gradient hypothesis as here formulated that the act associated with the shorter delay of reinforcement will acquire habit strength at a faster rate than will the alternative act. However, because of the principle of oscillation, the stronger of the two habits would be expected to attain dominance, not at once but only gradually, and often imperfectly, even after a very large number of trials. This *a priori* expectation is confirmed by experiment.

On the same principle it is to be expected that, other things equal, the larger the difference in the delays associated with the competing acts, the fewer will be the trials required to produce a given per cent of dominance. Similarly, for a given *difference* in

the delays associated with the respective acts, the longer the actual delays involved, the greater will be the number of trials required to produce a given per cent of dominance. Both these corollaries are also confirmed by experiment.

Perhaps the most critical test of the hypothesis concerns the ease of learning to discriminate comparable acts associated with delays of equal relative, but of different absolute, duration. The exponential goal gradient hypothesis as here formulated implies that the discriminability of acts associated with delays of reinforcement standing in the same ratio to each other will be maximal at a central region of absolute delays, but at other delays, whether greater or less, the ease of discrimination declines progressively. Moreover, it follows from these same principles that the point of maximum ease of discrimination of a coarse ratio, such as 1 to 3, will appear at smaller absolute values than will be the case with a fine ratio, such as 1 to 2. All of these intricate and detailed implications of the exponential goal gradient hypothesis are in remarkable agreement with the experimental observations at present available. This fact goes far to indicate that the goal gradient is a negative growth, or exponential, function. Incidentally this furnishes an excellent example of the manner in which indirect procedures may sometimes yield the characteristics of a function which has proved refractory to direct attack.

NOTES

Historical Note

The first mention of the gradient-of-reinforcement principle which we have been able to find was by Thorndike, in 1913. At that time he wrote: "Such intimacy, or closeness of connection between the satisfying state of affairs and the bond it affects, may be due to close temporal sequence. . . Other things being equal, the same degree of satisfyingness will act more strongly on a bond made two seconds previously than on one made two minutes previously. . ." (11, pp. 172-173). Thirteen years later, Margaret Washburn remarked in the third edition of *The Animal Mind*: "The facts show that it [the drive] will set most strongly in readiness those movements which most immediately preceded its resolution on a previous occasion. This 'gradient' of excitation from movements just before the final 'success,' step by step to those at the beginning of the series, may also be explained by the ordinary associative laws. The movements nearest the end of the series have a greater readiness due to recency of performance." (14, p. 335).

In 1932 the same general concept was put forward by the present writer in an attempt at a deductive explanation of numerous molar phenomena of animal

learning, such as the preference of the shorter path to a goal, the fact of blind alley elimination, the backward order of such eliminations, and so on: "The mechanism which in the present paper will be mainly depended upon as an explanatory and integrating principle is that the goal reaction gets conditioned the most strongly to the stimuli preceding it, and the other reactions of the behavior sequence get conditioned to their stimuli progressively weaker as they are more remote (in time or space) from the goal reaction. This principle is clearly that of a gradient, and the gradient is evidently somehow related to the goal. We shall accordingly call it the *goal gradient hypothesis*." (6, pp. 25-26.)

In order to perform these deductions it was necessary to postulate the mathematical characteristics of the gradient of habit strength which both Thorndike and Washburn had specified in a general way. Largely because of the results of Yoshioka's investigation (17), habit strength was postulated as being a function of the logarithm of the amount of time (t) or space separating the receptor-effector conjunction from the reinforcing state of affairs, i.e.,

$$sH_R = a - b \log t.$$

In 1938, however, the logarithmic gradient was rejected on the grounds (1) that when $t = 0$, sH_R becomes infinite, and (2) that with large values of t , sH_R becomes negative, both of which seem *a priori* improbable (7, p. 273). Accordingly the logarithmic equation was replaced by an exponential equation. In terms of our present notation this is:

$$m' = M'e^{-t}; \quad (13)$$

where M' is the learning asymptote under a given reinforcing agent, t has the same significance as in the logarithmic equation, and m' is the learning asymptote with a given delay in reinforcement and reinforcing agent. The exponential equation, because of the excellent agreement of its implications with a considerable range of empirical findings, is regarded as the closest approximation to the goal gradient function at present available.

The Wolfe Data

Fortunately, Wolfe (16) published the per cent of correct runs for each of his eight groups of rats at each of the ten days of the training process. An examination of these data suggested that the scores of days 7, 8, 9, and 10 are the most stable and significant of the series for our present purposes. Accordingly these probability-of-success scores for the four days were pooled for each group of

The next step was to convert these probability-of-success scores into units of amount on some linear scale. This was done on the assumption (see Chapter XIII) that learning manifests itself in this experiment only by overriding an oscillatory tendency varying from moment to moment, the net result of which, on the average, is to give about equal numbers of choices to the right and the left paths. This chance oscillation factor is assumed further to distribute itself approximately according to the Gaussian or "normal" law of chance. Convenient tables of this function have been derived by mathematicians, whereby any per cent of probability may be at once converted into amounts in presumably linear units. The unit in such cases is usually the standard deviation (σ) of the distribution of the chance variability involved. A simplified table of this kind

is shown as Table 9 on page 311. However, a more elaborate table was used with the present data. The use of the table may be illustrated as follows: the pooled scores of days 7, 8, 9, and 10 of the one-minute group show 64.9 per cent of correct reaction. Referring to our table we find that this corresponds to a shift .40 σ from the pure chance (50-50) distribution of choices in the correct direction. In this way were obtained the following σ -values which may be regarded as indices of habit strength:

<i>Amount of delay</i>	<i>Index of habit strength at trials 7, 8, 9, and 10</i>
0"	1.21 σ
5"	.98 σ
30"	.49 σ
1'	.40 σ
2.5'	.42 σ
5'	.28 σ
10'	.30 σ
20'	.18 σ

Figure 30 is plotted from these values. The negative growth function fitted to the first four of the values is:

$$m' = .375 + (1.21 - .375)e^{-.066t}$$

The smooth curved line of Figure 31 is plotted from this equation.

Some indirect confirmation of the hypothesis that more than one factor is operating to produce Wolfe's empirical gradient is furnished by the fact that it was found possible to get a rather satisfactory fit to the above set of values by assuming that they represent the simple summation of two growth functions, a major one corresponding roughly to the equation given above, and a minor one representing a rather slow learning process with a gentler slope. The complete equation, including both of the supposed components, is:

$$m' = .175 + .225e^{-.0017t} + .810e^{-.066t}$$

The Characteristics of Perin's Empirical Gradient

The curve which has been fitted to Perin's empirical gradient of reinforcement is a diminishing exponential function which has as its asymptote, not a horizontal straight line as have ordinary exponential functions, but a straight line which slopes downward in such a way as to cross the horizontal axis. This equation, shown graphically in Figure 32, is:

$$\begin{aligned} N &= 40 \\ \tan \Sigma \quad \Delta sH_R &= 1.6 \times 10^{-15}t - .043t + 1.45, \\ N &= 60 \end{aligned}$$

$$N = 40$$

in which $\tan \Sigma \quad \Delta sH_R$ represents the tangent of the curve of learning at the $N = 60$

point of 50 per cent choice of the act which removes the manipulative bar. This, it will be noticed, is by no means the same as habit strength (sH_R). Moreover, there is reason to believe that appreciable amounts of extinction effects con-

tribute to this function which may account for the fact that extrapolation where $t > 34$ yields negative values.

Does the Delay-of-Reinforcement Gradient Extend Forward as Well as Backward from the Point of Reinforcement?

Thorndike and his associates have reported evidence which suggests that the gradient of reinforcement may extend also in the forward direction; i.e., that learning may be reinforced when the reinforcing state of affairs *precedes* the $S - R$ conjunction as well as when it follows it. Recently Jenkins (8) has confirmed these results in a convincing manner, using albino rats as subjects in a maze situation. These studies indicate that the maximum of the forward gradient is considerably lower than that of the backward gradient. The adaptive significance of this second gradient has not as yet been very carefully studied. However, since the reduction in a need necessarily follows rather than precedes the act which brings this about, it would seem that the forward gradient could hardly play much rôle in selective learning.

Yoshioka's Study of Path-Length Discrimination in Rats

A number of years ago, Yoshioka (17) investigated experimentally the relative difficulty of setting up a consistent preference for the shorter of two paths to a food goal. In general he found that the ease of setting up the short-path preference was approximately the same for two mazes, one of which was twice as large as the other, for five different ratios of long to short path in each maze.

But how can Yoshioka's results be reconciled with the gradient taken as an exponential function of the form,

$$m' = M'e^{-at}$$

We have already shown above (p. 156) that if two pairs of delays in reinforcement, one pair twice as long as the other, are chosen at certain points at each side of a central point of maximum discriminability, one pair will be approximately as easy to learn as the other. It is evident that there may be found a very large number of such pairs of alternative delays. It is possible that Yoshioka chanced to select a series of pairs of alternate pathways involving just such delays in reinforcement.

Equations From Which the Various Theoretical Curves and Tables Have Been Derived

The exponential or negative growth function of the goal gradient from which Table 2 and Figure 33 were derived is,

$$m' = M' \times 10^{-.00672 t}, \quad (14)$$

where M' is the maximum strength attainable with an unlimited number of reinforcements with the reinforcing agent employed.

The equation from which Table 3 and the curves of Figure 34 were derived is,

$$sH_E = m' - m' \times 10^{-.02226 N},$$

where m' is as defined by the preceding equation.

The method of deriving the per cent of choices of the act associated with the shorter delay of reinforcement is first to calculate the strength of the respective

habits (H_1 and H_2) by means of equations 1 and 2; then substitute these values in equation 15:

$$H_1 - H_2 \quad (15)$$

where σ_1 is the standard deviation of the oscillation of H_1 , and σ_2 is the standard deviation of the oscillation of H_2 . Substitute the values of H_1 , H_2 , σ_1 , and σ_2 in this equation, solve for x , and look up the value of p , probability of occurrence or per cent of dominance of the stronger habit, in a table of the probability integral.

Example: Let us take the habit strengths yielded by delays of 30 and 60 seconds at 40 reinforcements (Table 3, columns 2 and 3). These turn out to be 43.8 habs and 27.5 habs respectively. Also it will be recalled that the oscillation range of both habits was assumed to have a standard deviation of 13 habs. Substituting these values in equation 3, we have,

$$\begin{aligned} x &= \frac{43.8 - 27.5}{\sqrt{13^2 + 13^2}} \\ &= \frac{16.3}{\sqrt{338}} \end{aligned}$$

$$\therefore x = .886.$$

Looking on page 76 of Kelley's *Statistical Tables* at $x = .886$, it is found that corresponding to this value is a p value of .812; i.e., the stronger of the two habits will dominate in the long run 81.2 per cent of the test trials, exactly as is shown at the bottom of column 5.

REFERENCES

1. ANDERSON, A. C. Time discrimination in the white rat. *J. Comp. Psychol.*, 1932, 13, 27-55.
2. ANDERSON, A. C. Runway time and the goal gradient. *J. Exper. Psychol.*, 1933, 16, 423-428.
3. DECAMP, J. E. Relative distance as a factor in the white rat's selection of a path. *Psychobiology*, 1920, 2, 245-253.
4. GRICE, G. R. An experimental study of the gradient of reinforcement in maze learning. *J. Exper. Psychol.*, 1942, 30, 475-489.
5. HAMILTON, E. L. The effect of delayed incentive on the hunger drive in the white rat. *Genet. Psychol. Monogr.*, 1929, 5, 131-207.
6. HULL, C. L. The goal gradient hypothesis and maze learning. *Psychol. Rev.*, 1932, 39, 25-43.
7. HULL, C. L. The goal gradient hypothesis applied to some 'field-force' problems in the behavior of young children. *Psychol. Rev.*, 1938, 45, 271-299.
8. JENKINS, W. O. Studies in the spread of effect. Ph.D. thesis, Yale University, 1942.
9. MILLER, N. E., and MILES, W. R. Effect of caffeine on the running speed of hungry, satiated, and frustrated rats. *J. Comp. Psychol.*, 1935, 29, 397-412.
10. PERIN, C. T. The effect of delayed reinforcement upon the differentiation of bar responses in white rats. *J. Exper. Psychol.*, 1943, 32, 95-109.
11. THORNDIKE, E. L. *Educational psychology*, Vol. I. *The original nature of man*. New York: Teachers College, Columbia Univ., 1913.

12. THORNDIKE, E. L. *The fundamentals of learning*. New York: Teachers College, Columbia Univ., 1932.
13. WARDEN, C. J., and HAAS, E. L. The effect of short intervals of delay in feeding upon speed of maze learning. *J. Comp. Psychol.*, 1927, 7, 107-116.
14. WASHBURN, M. F. *The animal mind* (third edition). New York: Macmillan, 1936.
15. WATSON, J. B. The effect of delayed feeding upon learning. *Psychobiology*, 1917, 1, 51-60.
16. WOLFE, J. B. The effect of delayed reward upon learning in the white rat. *J. Comp. Psychol.*, 1934, 17, 1-21.
17. YOSHIOKA, J. G. Weber's law in the discrimination of maze distance by the white rat. *Univ. Calif. Pub. in Psychol.*, 1929, 4, 155-184.

CHAPTER XI

Habit Strength as a Function of the Temporal Relation of the Conditioned Stimulus to the Reaction

Each of the last three chapters has been concerned with the quantitative aspects of one of the antecedent conditions of reinforcement which determine habit strength. In this, the fourth chapter on this general subject, we shall consider the functional dependence of habit strength upon a pair of closely related antecedent conditions. These have already been laid down (p. 71) as the necessary qualitative conditions for learning, namely, that there must be a temporal contiguity between an effector activity and (1) an afferent impulse or (2) the perseverative trace of such an impulse. We shall begin with the consideration of the former.

HABIT STRENGTH AS A FUNCTION OF THE DURATION OF THE CONDITIONED STIMULUS AT THE TIME OF REACTION OCCURRENCE

The question of the rate of habit formation as a function of the time the conditioned stimulus (\dot{S}) has been acting when the reaction (R) occurs has been submitted to systematic study by Kappauf and Schlosberg (5). These investigators delivered the unconditioned stimulus, in the form of a 1/3-second electric shock, to the right front leg of each of a series of albino rats. The conditioned stimulus was a loud buzzer which temporally overlapped the shock. With different groups of animals the buzzer began 1/3, 2/3, 1, 2, 4, and 7 seconds before the shock, both stimuli terminating at the same time. Accordingly the habits thus set up would, in Pavlov's terminology (6, p. 88), be called "delayed" conditioned reflexes, as contrasted with "trace" conditioned reflexes (6, p. 40) in which the action of the conditioned stimulus terminates before the onset of the unconditioned stimulus.

Kappauf and Schlosberg recorded and measured several different responses to shock which it was thought might become conditioned. Possibly because of the small number of animals employed in each group and the fact that each group was subdivided by differential treatment, the various reactions yielded somewhat dis-

cordant results. The single response which gave the most consistent conditioned reactions was a sharp inspiration or gasp. By pooling measurements taken from published graphs representing the scores of the two animals making up each of the six delay groups, there has been obtained an indication of what purports to be the functional relationship which we are seeking. The values so secured are represented by the circles of Figure 38.

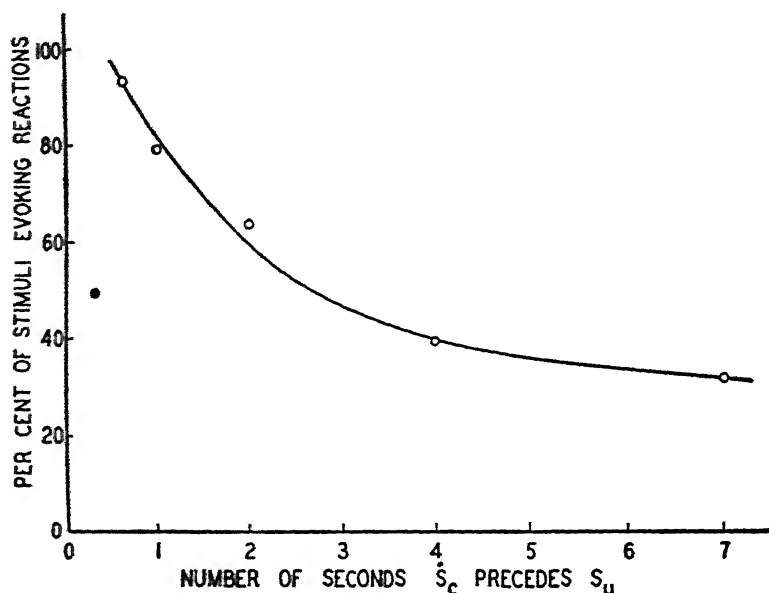


FIG. 38. Graphic representation of the per cent of conditioned stimuli evoking antedating reactions in a delayed conditioned reflex, as a function of the time interval from the beginning of the conditioned stimulus to the onset of the unconditioned stimulus. (Plotted from pooled measures of two graphs showing per cent of conditioned gasping reactions in rats, published by Kappauf and Schlosberg 5.)

It is at once evident from an inspection of the arrangement of these circles that long-delayed conditioned stimuli are much less effective in acquiring receptor-effector connections under reinforcement conditions than are those of relatively short delay. Moreover, beginning at the delay of maximum efficiency there appears to be a progressive falling off in the per cent of stimulations evoking conditioned reactions, the decline taking place approximately according to a simple negative growth or decay function of the delay beyond the optimum. Such a function was fitted to the values

corresponding to the circles, and is represented by the curve drawn among them. The asymptote or ultimate level of fall turns out to be 30.4 per cent. This suggests that generalized response sensitization due to shock was occurring in this experiment, or that an auditory stimulus will not wholly lose its capacity for becoming conditioned to a reaction however much it may be prolonged, or both.

Turning to the left-hand side of Figure 38, there is found a definite suggestion that learning is facilitated by having the unconditioned stimulus occur a fraction of a second later than the conditioned stimulus. The Kappauf-Schlosberg experimental technique makes difficult the distinction between conditioned and unconditioned reactions in this region; therefore the discussion of this point will be taken up in connection with an investigation presently to be considered, which does not suffer from such a handicap.

From the preceding analysis, then, we conclude that within the limits of the Kappauf-Schlosberg investigation:

1. The maximum efficiency of conditioning occurs when the onset of the unconditioned stimulus follows that of the conditioned stimulus by a fraction of a second.
2. As the delay in the onset of the unconditioned stimulus increases beyond that yielding the maximum learning efficiency, the rate of habit-strength acquisition decreases progressively according to a simple decay function of the amount of this additional delay.
3. The value of this function at the limit of its fall is about a third of that at its highest point. This asymptotic value presumably approximates the status in learning situations of static, i.e., non-changing, stimulus elements.

A NEUROLOGICAL HYPOTHESIS AND SOME COROLLARIES

The results shown in Figure 38 present such a striking parallel to Figures 4 and 5, particularly to Figure 4, that it has been considered worth while to give brief consideration to some implications of a related neurological hypothesis which has been suggested by Kappauf and Schlosberg (5, p. 39). The relevant neurological phenomena may be summarized briefly as follows:

1. Receptor discharge impulses begin an appreciable interval after the impact of the stimulus energy on the receptor (1, p. 116; 2).
2. As the energy impact on the receptor becomes weaker, the discharge latency becomes longer (see Figure 3).

3. Following the period of receptor-discharge latency there is a period of relatively rapid recruitment in the frequency of receptor discharge impulses, which usually reach their maximum within a second (1, p. 116).

4. The amount of stimulus energy ultimately applied remaining constant, the faster its rate of application, the more rapid will be the rate of recruitment and the greater the maximum frequency of receptor-discharge impulse (1, p. 75).

5. If the rate of stimulus impact is relatively abrupt and constant, the greater the stimulus energy applied, the greater will be the maximum frequency of receptor impulse discharge (1, p. 116).

6. Following the attainment of the maximum frequency of receptor discharge impulses, the stimulus meanwhile continuing to act unchanged, there ensues a progressive decline in frequency approximately according to a simple decay function. In some receptors, such as touch and those associated with hairs, the frequency quickly falls to zero; in others, such as pressure and those associated with muscle spindle, the frequency becomes constant at a level well above zero (1, p. 79).

7. In case the impact of the stimulus energy on the receptor organ ceases before the point of maximum receptor-discharge frequency is reached, there is usually a brief after-discharge which apparently may be prolonged under certain circumstances by self-propagating central processes (9). This latter perseverative activity presumably declines as a simple negative growth function of the time since stimulus termination, the asymptote of this decline being zero (Figure 5, p. 43).

We now add to this summary of empirical findings a formalized statement of Kappauf and Schlosberg's hypothesis¹: *Other things equal, the increment to the strength of a receptor-effector connection (ΔH_r) resulting from a reinforcement is an increasing function of the frequency of the associated receptor discharge, or the intensity of the resulting afferent impulse.*

Our immediate concern here is with the implications of the Kappauf-Schlosberg hypothesis and certain items of the receptor impulse summary presented above, namely, items 3, 4, 5, 6, and 7.

I. It follows from the above hypothesis and empirical item 3 that in a reinforcement situation there is a temporal relationship of the conditioned to the unconditioned stimulus such that as the onset of the unconditioned stimulus is progressively delayed, the rate of learning will increase.

II. It follows from the hypothesis and empirical item 4, other factors remaining constant, that in a reinforcement situation the

¹ Kappauf and Schlosberg are in no way to be held accountable for the shortcomings of this formulation; the present author assumes entire responsibility.

slower the rate of application of the conditioned stimulus energy, the slower will be the rate of habit-strength acquisition.

III. It follows from the hypothesis and empirical item 5 that *in a reinforcement situation, temporal relationships of conditioned and unconditioned stimuli remaining optimal, within moderate ranges the greater the conditioned stimulus energy applied, the more rapid will be the rate of habit-strength acquisition.*

IV. It follows from the hypothesis and empirical item 6 that *in a reinforcement situation as the time of the onset of the unconditioned stimulus is further retarded beyond the point of optimal timing, the rate of learning will decline, but at a rate slower than the rate of rise during the recruitment period, the course of the decline following a negative growth function of the amount of delay, with asymptote appreciably above zero in the case of certain receptors.*

V. It follows from the hypothesis and empirical item 7 that *in a reinforcement situation as the time of the onset of the unconditioned stimulus is retarded beyond the optimal amount, the action of the conditioned stimulus having ceased before the maximum rate of receptor discharge impulse is reached, trace conditioned reactions will be generated.* In such cases the rate of learning will decline according to a simple negative growth function of the amount of delay, with its asymptote at zero.

We may now briefly compare the above deductions with the facts of habit-strength acquisition. Corollary III is in agreement with the empirical observations as reported by Pavlov (6). As yet no evidence has been found concerning Corollary II. Corollaries I and IV are in good qualitative agreement with the empirical results of Kappauf and Schlosberg. At the best the above deductions may constitute the beginning of a passage of the molar theory of behavior over into the ultimate molecular behavior theory based on neurophysiology; at the worst they may be no more than a harmless failure in the long trial-and-error history which must precede the evolution of a true molecular theory of learned behavior.

HABIT STRENGTH AS A FUNCTION OF THE DURATION OF THE STIMULUS TRACE AT THE TIME OF ACTION OCCURRENCE

The fifth corollary derived from the Kappauf and Schlosberg hypothesis, since it concerns the rate of learning as a function of

the age of the stimulus trace which is contiguous with the reaction to be associated in the learning process, leads directly to the second factor whose relation to rate of habit-strength acquisition is to be considered in the present chapter. A study bearing directly on this point has been published by Helen Morrill Wolfe.

In one of her two experiments in this field (12, 13), Mrs. Wolfe employed 90 human subjects in groups of ten, each group devoted to the determination of the effectiveness of trace conditioned-reflex learning with a particular temporal relationship of the conditioned

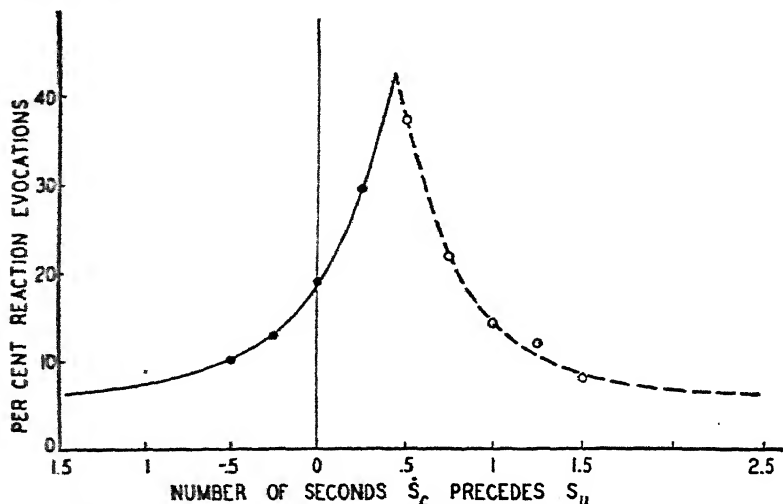


FIG. 39. Graphic representation of habit strength as a function of the temporal relation of the conditioned to the unconditioned stimulus in a "short" trace conditioned reaction. Both curves are simple negative growth functions whose asymptotes are 6.5. (From data published by Helen Morrill Wolfe, 12.)

to the unconditioned stimulus. The former was a single sharp click, the latter, an electric shock to the hand. The reactions recorded were hand-withdrawal movements associated with shock avoidance. At irregular intervals throughout the reinforcement process the conditioned stimulus was presented without the accompanying shock. The measure of learning was the per cent of hand movements following these presentations.

The results of this investigation are shown by the series of circles in Figure 39. A glance at these circles suggests a confirmation of Corollary I, the maximum efficiency appearing when the

shock followed the click by a fraction of a second. This we shall call the point of *optimal stimulus asynchronism*. In this the trace conditioned reaction of Mrs. Wolfe agrees substantially with the delayed conditioned reaction of Kappauf and Schlosberg.

Corollary V is concerned with the hollow circles, which represent learning efficiency when the shock, and so the reaction and its reinforcement, followed the click by a half second or more. An examination of this portion of Figure 39 reveals that as the onset of the shock is retarded beyond the point of optimal stimulus asynchronism the habit strength diminishes consistently, the rate of diminution decreasing as zero efficiency is approached, quite in agreement with the corollary. The progressive decline in habit strength on this side of the point of optimal learning efficiency we shall call the *posterior stimulus asynchronism gradient*.

In order to test the corollary in more detail a simple negative growth function was fitted to these latter values; this is represented by the broken line passing among the hollow circles. Notwithstanding the usual deviations, the circles fall fairly close to the line, which indicates a reasonably good fit. The limit of fall of this function turns out to be 6.5, a value appreciably above zero. In this respect empirical results appear to disagree with Corollary V; however, the failure of this stimulus-asynchronism gradient to fall to zero may be due to sensitization effects, i.e., the effects of the shock alone quite apart from its association with the conditioned stimulus (4, p. 431).

THE PROBLEM OF BACKWARD CONDITIONING

"Backward" conditioning is said to take place where the stimulus originally evoking the reaction, and usually the reaction itself, occur before the impact of the conditioned stimulus. This order of occurrence during the reinforcement process is called *backward* because it is the reverse of the order of occurrence when the acquired receptor-effector connection functions; in the latter situation, of course, the stimulus must precede the reaction it evokes.

At one time Pavlov (6, p. 27) regarded backward conditioning as impossible; later he revised this opinion (7, p. 381), holding that while backward conditioning is possible, the results obtainable by this procedure are very weak and unstable. Upon the whole the latter view has been substantiated by more recent studies (9), including two experiments by Mrs. Wolfe (12, 13).

The two extreme left-hand circles of Figures 39 represent the results attained by backward conditioning. In this connection it may be noted that these two circles occupy a position on a smooth and consistent gradient descending from a point near that of maximum learning efficiency. We shall call this the *anterior stimulus-asynchronism gradient*. This continuity suggests that so-called "backward" conditioning may be physiologically no more than a special and extreme case of a gradient antedating not the onset of the conditioned stimulus but, rather, the optimal phase of stimulus asynchronism. In order to test the hypothesis a simple negative growth function was fitted to the four values represented by the four solid circles. This is shown graphically by the smooth curve drawn through these circles; the fit may be seen to be nearly perfect. Whether or not it be a coincidence, the asymptote of this gradient is exactly the same as that of the values at the right, namely, 6.5 per cent. As in the case of the posterior stimulus asynchronism gradient, the small positive asymptotic value may very well be due to sensitization effects (4, p. 831).

As a final word concerning the data represented in Figure 39 it may be added that the two gradients were extrapolated upward to where they intersect, on the assumption that the point of intersection would indicate indirectly somewhat more precisely the optimal temporal relationship of the conditioned to the unconditioned stimulus. The outcome of this manoeuvre is shown graphically in Figure 39. It suggests that under the conditions of Mrs. Wolfe's experiment the conditioned stimulus should antedate the onset of the unconditioned stimulus by .44 second if maximum learning efficiency is to be attained. It also suggests that had a group of subjects been conditioned at this interval the learning yield would have been 42.4 per cent of reaction evocations, an appreciable advantage over the value obtained at a delay of .5 second.

The results of the above analysis are in agreement with a considerable body of experimental evidence which indicates that for optimal learning to take place the conditioned stimulus should precede the onset of the unconditioned stimulus by something less than a half second. A certain amount of speculation has arisen regarding the cause of this now well-established fact. One of the most ingenious of these hypotheses has been put forward by Guthrie (3), who has suggested that in the learning situation the stim-

ulus actually conditioned to the reaction is the proprioceptive stimulation arising from reactions, usually implicit, evoked by the conditioned stimulus at the outset of the learning. This obviously *ad hoc* hypothesis accounts for the optimal time relationship of the onset of conditioned and unconditioned stimulation, though it fails to explain why proprioceptive stimuli should have a monopoly in the acquisition of receptor-effector connections over discharges from other important receptors such as the ear and the eye. It seems likely that the solution of the problem must await the future developments of neurophysiology; fortunately this is not a necessary prerequisite to the development of a molar system of behavior theory.

"SHORT" VERSUS "LONG" TRACE CONDITIONED REACTIONS

Intimately related to the gradient of habit strength as a function of the age of the trace at the time of reaction occurrence is Pavlov's distinction between short and long trace conditioned reflexes. In this connection Pavlov remarks (6, p. 40):

Trace reflexes may be of different character, depending on the length of pause between the termination of the conditioned stimulus and the appearance of the unconditioned stimulus. When the pause is short, being a matter of only a few seconds, then the trace left by the conditioned stimulus is still fresh, and the reflex is what we may term a *short-trace reflex*. On the other hand, if a considerable interval, one minute or more, is allowed to elapse between the termination of the conditioned and the beginning of the unconditioned stimulus we have a *long-trace reflex*. . . every stimulus must leave a trace on the nervous system for a greater or less time—a fact which has long been recognized in physiology under the name of after effect.

The referent of the term *trace* in the last sentence quoted above is evidently the same as that of the expression *perseverative stimulus trace* in the present work. We thus arrive at the identification of Mrs. Wolfe's right-hand gradient (Figure 39) with Pavlov's "short" trace conditioned reflex.

Pavlov is less specific about the basis for his "long" trace conditioned reflexes, though his experimental examples and associated remarks make the picture fairly clear (6, pp. 41-42):

This may be illustrated by the following detailed experiment of Dr. Feokritova: A dog is placed in a stand and given food regularly every thirtieth minute. In the control experiments any-one feeding after the

first few is omitted, and it is found that despite the omission a secretion of saliva with a corresponding alimentary motor reaction is produced at about the thirtieth minute. Sometimes this reaction occurs exactly at the thirtieth minute, but it may be one or two minutes late. In the interval there is not the least sign of any alimentary reaction, especially if the routine has been repeated a good number of times. When we come to seek an interpretation of these results, it seems pretty evident that the duration of time has acquired the properties of a conditioned stimulus.

What is the physiological meaning of these time intervals in their rôle as conditioned stimuli? . . . Time is measured from a general point of view by registering different cyclic phenomena in nature, such for instance as the rising and setting of the sun or the vibration of the pendulum of a clock. But many cyclic phenomena take place inside the animal's body.

In a word, the explanation of the striking outcome of Dr. Feokritova's experiment seems to be that each feeding of the dog initiated the stable internal cycle of digestion which activated somewhat different receptors at each of its phases. The receptor discharges released by the phase reached after 30 minutes of digestion were naturally conditioned to the salivary process evoked and reinforced by each subsequent feeding. The afferent process conditioned under such circumstances would not be a perseverative stimulus trace but, rather, the afferent impulse arising directly from receptor discharges. For this reason considerable confusion might be avoided if reactions conditioned directly to stimuli arising from such physiological cycles were called *cyclic-phase conditioned reactions*.

A cyclic-phase conditioned reaction involving another type of physiological cycle may easily be set up (4, pp. 417-418). An electric shock may be delivered to a subject 30 times at regular intervals of a half minute. Each shock will cause the subject to react rather strongly, releasing various endocrine secretions and otherwise upsetting his equilibrium. Presumably the body at once will begin to shift back to normal in much the same way after each shock. Presumably also, each phase of this recovery cycle will activate a somewhat different set of receptors. Thus just before the onset and cessation of each shock the same set of receptors will be discharging as on the previous occasions and so will become conditioned to the reactions evoked by the shock, e.g., the *galvanic skin reaction*. It naturally follows that if the shock is omitted the discharge of these receptors will evoke the reaction at about its usual time of incidence, much as if the shock were deliv-

ered. A record of such a cyclic-phase conditioned reaction is reproduced as Figure 40.¹

Let it be supposed that a stimulus incidentally becomes conditioned to a strong reaction, such as the one to shock, which involves a certain time for the return of the body to equilibrium.

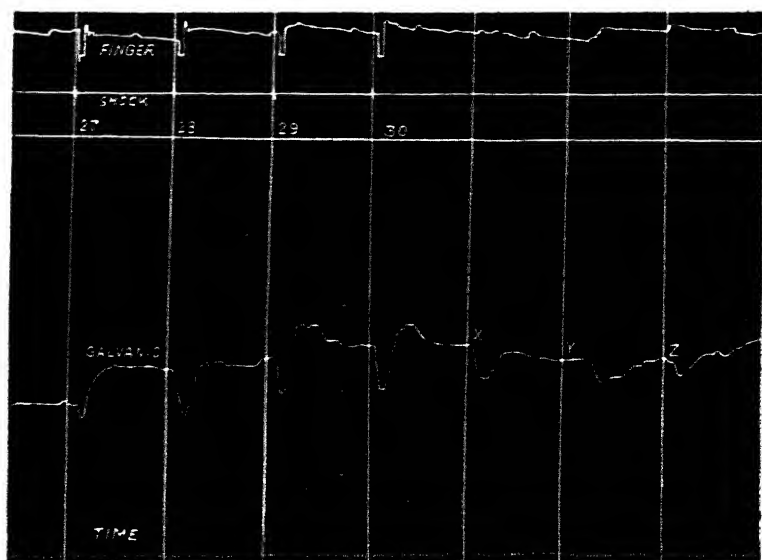


FIG. 40. Reproduction of a record showing a cyclic-phase conditioned reaction in a human subject. The tracing shows the reactions to the last four of 30 induction shocks delivered at 38.5-second intervals. Presumptive conditioned galvanic reactions to the temporal interval appear at X, Y, and Z. The vertical lines have been drawn to show points of simultaneity on the several tracings. (Reproduced from 4, p. 418.)

Such a stimulus will necessarily evoke the reaction during the learning process, thereby initiating an internal behavior cycle which may be some minutes in length. A conditioned stimulus combination of this nature would yield a conditionable process which would extend far beyond the range of a true perseverative

¹ Under the author's direction, Mr. R. O. Rouse performed a modified form of this experiment, in which a shock was delivered every 30 seconds. Seven of eleven subjects displayed temporal conditioning in one form or another analogous to that shown in Figure 40, some giving evidence of anticipatory or anxiety tendencies. One subject who gave clear indications of "temporal" conditioning admitted, upon subsequent questioning, that he had counted; none of the other subjects reported having done this.

receptor discharge, and might easily give rise to what would superficially appear to be trace conditioned reactions in which the stimuli would be separated by many times three seconds. This presumptive mechanism may explain the paradox that certain studies, such as those of Warner (11) and Yarbrough (14), purport to show reactions conditioned to perseverative stimulus traces in which the supposed "trace" at the point conditioned may be as old as twenty seconds.

SUMMARY

Numerous experiments have shown that, the gradient of reinforcement remaining constant, the most favorable temporal arrangement for the delivery of the conditioned and the unconditioned stimuli is to have the latter follow the former by something less than a half second. But as the asynchronism of the onset of the two stimuli deviates from this optimal relationship in either direction there is a falling off in the habit strength which will result from a given quality and number of reinforcements, the rate of decline in each direction probably being a simple negative growth or decay function of the nature and extent of stimulus asynchronism. The situation where the onset of the unconditioned stimulus, as well as the reaction and its reinforcement, antedates the optimal relationship by more than a half second or so includes two special cases which are traditionally known as *simultaneous* conditioning and *backward* conditioning. These are believed to be portions of the same physiological continuum antedating the point of optimal stimulus asynchronism. This yields the anterior stimulus-asynchronism gradient.

The case where the unconditioned stimulus falls later than the optimal relationship has been studied somewhat more than the one in which it precedes this point. This develops two posterior gradients of stimulus asynchronism, depending on whether the conditioned stimulus terminates early or continues on to overlap the unconditioned stimulus. In both events, habit strength declines markedly as the delay in the onset of the unconditioned stimulus increases beyond the optimum, the diminution being a simple decay function of the amount of delay beyond the optimal relationship. If the conditioned stimulus persists, the resulting habit is said to be a *delayed conditioned reaction*. The rate of fall in habit strength as a function of increased asynchronism is moderate, and

the limit of the decline probably has a value considerably above zero. This level is believed to represent the status of static elements in stimulus complexes.

In case the conditioned stimulus is instantaneous the resulting habit is said to be a *trace conditioned reaction*, the rate of fall in habit strength is relatively great, and the limit of decline in the gradient when experimental artifacts are eliminated probably is zero. It is doubtful if true trace conditioned reflexes can be set up when the onset of the unconditioned stimulus follows the termination of the conditioned stimulus by more than about three seconds.

Both posterior-asynchronism gradients are tentatively regarded physiologically as increasing functions of the magnitude or intensity of the temporally contiguous afferent discharges. These in their turn are believed to be increasing functions of the frequency of impulses given off by the receptors. The gradient of afferent discharge intensity in the case of delayed conditioned reactions is supposed to arise from the continuous action of the conditioned stimulus upon its receptor; that in the case of trace conditioned reactions is thought to be a mere perseveration or trace of the afferent action after the conditioned stimulus which originally initiated it has ceased to act on its receptor. In the case of the continued action of a stimulus energy on a receptor, the \dot{s} presumably consists of the afferent discharge arising in the receptor at a given instant, plus the perseverative traces arising from the stimulation during preceding instants.

The *cyclic-phase conditioned reaction* superficially resembles the true trace conditioned reaction. In such situations the "stimulation" involves, in addition to a mere receptor discharge, the setting in motion of a major physiological cycle such as that of digestion or the return to equilibrium after an electric shock. If the stimuli activated by a particular phase of such a cycle are regularly conditioned to the reaction in question, this reaction will later be evoked at that phase of the cycle, and consequently at a certain time following the onset of a stimulus associated with the initiation of the cycle. Stimuli evidently originating in the digestive cycle in dogs have evoked conditioned reactions as much as half an hour after the last preceding significant stimulation. The results of such "temporal" conditioning have somewhat misleadingly been said to yield "long" trace conditioned reactions.

The considerations put forward in Chapters VI, VII, VIII, IX,

X, and the present one enable us to formulate our fourth primary principle, law, or postulate:

POSTULATE 4

Whenever an effector activity ($r \rightarrow R$) and a receptor activity ($S \rightarrow s$) occur in close temporal contiguity (sC_r), and this sC_r is closely associated with the diminution of a need (G) or with a stimulus which has been closely and consistently associated with the diminution of a need (\hat{G}), there will result an increment to a tendency (ΔsH_R) for that afferent impulse on later occasions to evoke that reaction. The increments from successive reinforcements summate in a manner which yields a combined habit strength (sH_R) which is a simple positive growth function of the number of reinforcements (N). The upper limit (m) of this curve of learning is the product of (1) a positive growth function of the magnitude of need reduction which is involved in primary, or which is associated with secondary, reinforcement; (2) a negative function of the delay (t) in reinforcement; and (3) (a) a negative growth function of the degree of asynchronism (t') of \hat{S} and R when both are of brief duration, or (b), in case the action of \hat{S} is prolonged so as to overlap the beginning of R , a negative growth function of the duration (t'') of the continuous action of \hat{S} on the receptor when R begins.

NOTES

Mathematical Statement of Postulate 4

The mathematical statement of Postulate 4 is distinctly more concise, convenient, and informative than is the verbal formulation given above. It has several cases depending on whether (1) \hat{S} is prolonged and overlaps the beginning of R temporally, and (2) their degree of asynchronism in case they are of brief duration and do not overlap temporally. As an illustration of the case where R follows and overlaps the continuous action of \hat{S} on the receptor for duration t'' , we have the following equation:

$$sH_R = M (1 - e^{-kw})e^{-kt}e^{-wt'} (1 - e^{-tN}), \quad (16)$$

where,

M = 100 habs, the physiological maximum of habit strength;

e = a mathematical constant usually taken in the present work as 10;

w = a constant change in a measurable objective criterion which results in a need reduction,

t = the delay in reinforcement;

$t' = T_R - T_{\hat{S}} - .66$, where \hat{S} is of more than instantaneous duration and overlaps the beginning of R ;

T_R = the time of the beginning of R ;

T_S = the time of the beginning of \dot{S} ;

N = the number of reinforcements;

k, j, u , and i = empirical constants.

The meaning of equation 16 may be clarified by the following example. Let it be supposed that we have a simple learning situation in which 20 reinforcements are given ($N = 20$); that 5 grams of a standard food are given a canine subject at each reinforcement ($u = 5$); that the reinforcement is given 3 seconds after the C ($t = 3$); and that \dot{S} begins a continuous action on the receptor 2 seconds before the beginning of R ($t'' = 2$). Taking the values of k, j, u , and i from previously given equations (11, 14, 20, and 6) fitted to empirical data, and substituting, we have,

$$sH_R = 100(1 - 10^{-.153 \times 5})10^{-.00672 \times 3} \times 10^{-.2515(2-.66)}(1 - 10^{-.013 \times 20}). \quad (17)$$

Solving by easy stages, we have,

$$\begin{aligned} sH_R &= 100\left(1 - \frac{1}{5.281}\right) \times \frac{1}{1.0475} \times \frac{1}{2.1725} (1 - 10^{-.013 \times 20}) \\ &= 100(.3562)(1 - 10^{-.013 \times 20}) \\ &= 35.62(1 - 10^{-.013 \times 20}), \end{aligned}$$

which presents the familiar equation expressing the positive growth curve of simple learning. Solving further, we have,

$$\begin{aligned} sH_R &= 35.62 \times .5635 \\ &= 20.1 \text{ habs.} \end{aligned}$$

In case the learning situation is the same as above except that both \dot{S} and R are of brief duration, \dot{S} preceding R by .9 second, the equation becomes:

$$\begin{aligned} sH_R &= 10(1 - 10^{-.153 \times 5})10^{-.00672 \times 3} \times 10^{-1.182(.9-.44)}(1 - 10^{-.013 \times 20}). \quad (18) \\ \therefore sH_R &= 12.5 \text{ habs.} \end{aligned}$$

In case the learning situation is the same as above except that R precedes S by one-tenth of a second, we have:

$$\begin{aligned} sH_R &= 10(1 - 10^{-.153 \times 5})10^{-.00672 \times 3} \times 10^{+1.068(-1-.44)}(1 - 10^{-.013 \times 20}). \quad (19) \\ \therefore sH_R &= 11.0 \text{ habs.} \end{aligned}$$

The Equation of the Curve of Figure 38

The negative growth function fitted to the values represented by the hollow circles of Figure 38 is:

$$y = 62.6 \times 10^{-.2515(x-1)} + 30.4 \quad (20)$$

where y is the per cent of antedating reactions and x is the time from the beginning of the conditioned stimulus to that of the unconditioned stimulus, the reaction, and the reinforcement. The number 30.4 represents the asymptote.

The Equations of the Curves of Figure 39

The negative growth function fitted to the hollow circles at the right of Figure 39 is:

$$y = 30.5 \times 10^{-1.182(x-.5)} + 6.5 \quad (21)$$

where y is the per cent or probability of conditioned-reaction evocation on the test trials and x is the time at which the reaction occurs (T_R) less the time at which the stimulus occurs (T_S); in case R precedes S , x will, of course, be negative. The number 6.5 represents the asymptote.

The negative growth function fitted to the values represented by the solid circles at the left side of Figure 39 is:

$$y = 22.5 \times 10^{-1.068(.25-x)} + 6.5, \quad (22)$$

in which y and the value 6.5 mean the same as in the equation fitted to the right-hand gradient, and the values of x must be less than .44 second.

Probably a somewhat more general and significant manner of writing the above equations is as follows:

$$p = 35.9 \times 10^{-x'} - 6.5, \quad (23)$$

in which,

p = the per cent or probability of conditioned-reaction evocation;

$t' = T_R - T_S - .44$;

T_R = the time of the instantaneous occurrence of R in seconds;

T_S = the time of the instantaneous occurrence of S in seconds;

$x' = -1.068$ if t' is negative, but $+1.182$ if t' is not negative.

Ultimately of course, the values of p in equations 20, 21, 22, and 23 will need to be converted into units of amount on the basis of the normal probability function (see p. 311 ff.), which will presumably change the equations in question as well as the forms of Figures 38 and 39. Despite this defect it is believed that these equations and figures have a certain amount of expository value as well as suggestive significance for further developments.

The Stimulus-Asynchronism Gradients and the Parameters of the Curve of Habit Acquisition

The question of the relation of the three stimulus-asynchronism gradients of conditioning to the slope and asymptote of the curve of habit-strength acquisition as a function of the number of reinforcements, arises here just as we have seen parallel questions arise in connection with the amount of the reinforcing agent employed and the gradient of reinforcement. It is clear that a satisfactory theory of learning and of learned behavior requires a knowledge of this relationship in all the cases mentioned. Unfortunately no relevant evidence has been found which is sufficient to yield a decisive indication as to the relationship in the case of the stimulus-asynchronism gradients. However, largely in order to pose the question before students of behavior principles, we postulate that it is one of several determinants of the asymptote of habit strength (m) with unlimited reinforcements.

The Derivation of the Equation Expressing Postulate 4

It is assumed that,

$$M' = M(1 - e^{-kw}), \quad (12)$$

$$m' = M'e^{-it}, \quad (14)$$

either

$$m = m'e^{-it'} \quad (24)$$

where (20)

$$t' = T_R - T_S - .66$$

or (23)

$$m = m'e^{-it''} \quad (25)$$

and

$$sH_R = m(1 - 10^{-iN}) \quad (26)$$

where M , M' , m' , and m are respectively: the absolute upper physiological limit of habit strength with unlimited reinforcement, the upper limit of habit strength as determined by the nature and amount of the reinforcing agent employed, the limit of habit strength as determined by the delay in reinforcement, and the limit of habit strength as determined by the degree of receptor-effector asynchronism.

Now, substituting equation 24 (or 25) in equation 26, we have,

$$sH_R = m'e^{-it'}(1 - 10^{-iN}) \quad (27)$$

Substituting equation 14 in equation 27, we have,

$$sH_R = M'e^{-it'}e^{-it''}(1 - 10^{-iN}) \quad (28)$$

Substituting equation 12 in equation 28, and recalling that $M = 100$ habs, we have,

$$sH_R = 100(1 - e^{-kw})e^{-it'}e^{-it''}(1 - 10^{-iN}),$$

which is one of the alternative equations sought (16).

It is altogether probable that there are other important factors which enter into the determination of habit strength in simple learning or conditioning situations. Two of these are the intensity of the conditioned stimulus (S) and the vigor or intensity of the reaction (R). Specific researches analogous to the studies of Gantt, Perin, Williams, Hovland, and others cited in the preceding pages need to be performed on these probable factors before their relationship to habit strength can be postulated with much confidence. In the end a grand investigation involving the finding of the joint reaction potentiality of *all* the presumptive determining factors when taken in all combinations of the representative values of each (after the manner of Perin's study of habit strength and motivation, δ) must be carried out before a really dependable equation for Postulate 4 can be written. The point is that habit strength as a function of t when w and t' are constant at a certain value, is not necessarily the same as it will be when w and t' are constant at other values. This will be a huge task, but the outcome should be worth the labor involved. It seems unlikely that the Fisher-design type of experiment will yield dependable indications of the complex hyperspatial curvatures which almost certainly will be found. Meanwhile, the equations given above may serve as points of departure for further empirical analyses.

REFERENCES

1. ADRIAN, E. D. *The basis of sensation*. New York: W. W. Norton and Co., 1928.
2. GRAHAM, C. H. Vision: III. Some neural correlations. Chapter 15 in *Handbook of general experimental psychology*, C. Murchison, editor. Worcester, Mass.: Clark Univ. Press, 1934.
3. GUTHRIE, E. R. Conditioning as a principle of learning. *Psychol. Rev.*, 1930, 37, 412-428.
4. HULL, C. L. Learning: II. The factor of the conditioned reflex. Chapter 9 in *Handbook of general experimental psychology*, C. Murchison, editor. Worcester, Mass.: Clark Univ. Press, 1934.
5. KAPPAUF, W. E., and SCHLOSBERG, H. Conditioned responses in the white rat. III. Conditioning as a function of the length of the period of delay. *J. Genet. Psychol.*, 1937, 50, 27-45.
6. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
7. PAVLOV, I. P. *Lectures on conditioned reflexes* (trans. by W. H. Gantt). New York: International Publishers, 1928.
8. PERIN, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. *J. Exper. Psychol.*, 1942, 30, 93-113.
9. ROSENBLUTH, A. Central excitation and inhibition in reflex changes of heart rate. *Amer. J. Physiol.*, 1934, 107, 293-304.
10. SWITZER, S. A. Backward conditioning of the lid reflex. *J. Exper. Psychol.*, 1930, 13, 76-97.
11. WARNER, L. H. The association span of the white rat. *J. Genet. Psychol.*, 1932, 41, 57-90.
12. WOLFLE, H. M. Time factors in conditioning finger-withdrawal. *J. Gen. Psychol.*, 1930, 4, 372-379.
13. WOLFLE, H. M. Conditioning as a function of the interval between the conditioned and the original stimulus. *J. Gen. Psychol.*, 1932, 7, 80-103.
14. YARBROUGH, J. U. The influence of the time interval upon the rate of learning in the white rat. *Psychol. Monog.*, 1921, 30, No. 135.

CHAPTER XII

Stimulus Generalization

The preceding chapters have shown that learning takes place according to various principles of reinforcement. In giving this account we have followed the conventional practice of characterizing learning as the setting up of receptor-effector connections. Moreover, we have represented these connections by such symbols as sH_R , sH_r , and $S \longrightarrow s \dashrightarrow r \longrightarrow R$, which specify only the receptor and effector processes actually involved in the reinforcement. It is now necessary to point out that while there is every reason to believe that each reinforcement does result in the connection represented by the symbolism, the actual outcome is much more complex than this. The fact is that every reinforcement mediates connections between a very great number of receptor and effector processes in addition to those involved in the reinforcement process and represented in the conventional symbolism sH_R . Several groups of such additional and indirectly established receptor-effector connections may be distinguished:

1. The reaction involved in the original conditioning becomes connected with a considerable zone of stimuli other than, but adjacent to, the stimulus conventionally involved in the original conditioning; this is called *stimulus generalization*.
2. The stimulus involved in the original conditioning becomes connected with a considerable zone of reactions other than, but related to, the reaction conventionally involved in the original reinforcement; this may be called *response generalization*.¹
3. Stimuli not involved in the original reinforcement but lying in a zone related to it become connected with reactions not involved in the original reinforcement but lying in a zone related to it; this may be called *stimulus-response generalization*.

The present chapter is particularly concerned with certain phenomena of stimulus generalization and some of their implications concerning adaptive behavior.

¹ The present analysis indicates that response generalization is a rather complex secondary phenomenon; space is not available in the present work for an adequate treatment of it.

PRIMARY STIMULUS-QUALITY GENERALIZATION

The molar principle of primary stimulus generalization is now well established both qualitatively and quantitatively, mainly by conditioned-reaction experiments. A few of the indirectly acquired

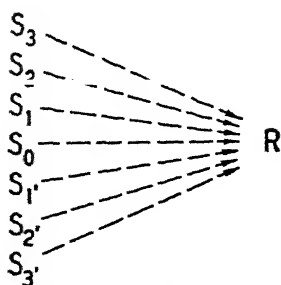


FIG. 41. Diagram of receptor-effector convergence arising from the primary stimulus generalization set up concurrently with the conditioning of $^H R$. S_1 , S_2 , S_3 represent positions at progressively greater distances on one side of S_0 on a one-dimensional stimulus continuum, and S_1' , S_2' , S_3' represent corresponding positions on the other side of S_0 on the same stimulus continuum. In this notation, \hat{S}_0 represents the stimulus when considered as involved in the reinforcement process, and S_0 , S_1 , S_2 , S_3 , etc., stimuli when considered as evoking reaction. S_0 and \hat{S}_0 are understood to fall at the same point on the stimulus continuum.

receptor-effector connections set up by the conditioning of S_0 to R are represented in Figure 41 as originating in the S 's with integral subscripts. Since all of these potentialities of reaction evocation converge from different stimulus possibilities upon the same reaction, stimulus generalization may be said to generate a *receptor-effector convergence*.

Under certain circumstances, e.g., in long trace conditioned reactions, generalization normally extends into receptor modes other than that involved in the reinforcement. Thus Pavlov reports the case of a defensive salivary reaction in a dog conditioned to the trace of a tactile stimulus, the reaction in question subsequently being evoked by a thermal stimulus of 0° centigrade (11, p. 113). It is important to note that while primary stimulus generalization may pass the boundaries of a given sense mode, this is not usual. Stimuli conditioned to one sense mode will ordinarily generalize only to other stimuli in the same sense mode, e.g., from one auditory vibration rate or intensity to another, or from one light wave length or intensity to another.

In general the more remote on the stimulus continuum the evoking stimulus (S) is from that originally conditioned (\hat{S}), the weaker will be the reaction tendency mobilized by it.

The quantitative law of primary stimulus generalization is nicely illustrated by an experiment reported by Hovland (4). This investigator conditioned the galvanic skin reaction in human subjects to a pure tone, e.g., of 1,967 cycles per second, and then meas-

ured the amplitude of the reaction evoked at this pitch and at three other pitches separated from each other by an equal number of discrimination thresholds (25 j.n.d.'s). The pooled results from twenty subjects are shown in Figure 42. There it may be seen that:

1. The amplitude of galvanic skin reaction diminishes steadily with the increase in the extent of deviation (d) of the evocation stimulus (S) from the stimulus originally conditioned (\bar{S}).

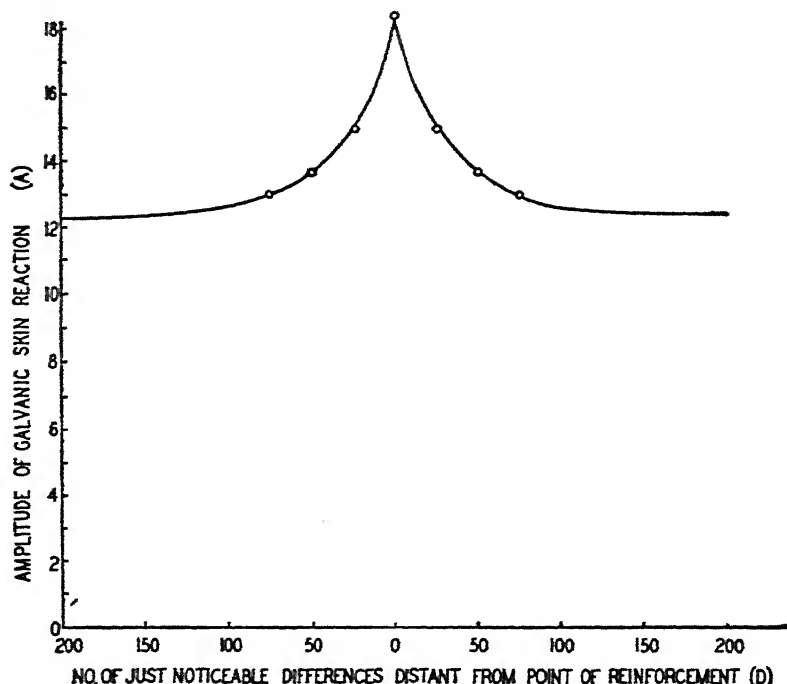


FIG. 42. Empirical generalization gradient of conditioned galvanic skin reaction derived from data published by Hovland (4). Note that the gradient extends in both directions on the stimulus continuum (vibration rate) from the point originally conditioned.

2. This diminishing generalization tendency extends symmetrically in both directions along the stimulus dimension.

3. The quantitative course of the diminution in the generalization tendency approximates rather closely a negative growth function of the amount (d) that S deviates from \bar{S} as measured in discrimination thresholds (j.n.d.'s). This is attested by the closeness with which the smooth curves, representing a simple decay function fitted to the generalization data, approximate the circles of Figure 42.

4. The asymptote or lower limit of the generalization gradient falls at 12.3 millimeters, rather than at zero. The failure of the gradient to approach zero as a limit is regarded as an experimental artifact due in part to the fact that previous to conditioning this reaction is evokable in appreciable amounts by any stimulus of even moderate intensity, in part to sensitization, and in part to the reaction becoming conditioned somewhat to the static stimuli arising from the experimental environment. The environmental portion of the stimulus situation, of course, remains constant throughout the changes in the tonal stimulus, which alone produce the gradient. Accordingly it is concluded that the asymptote of the true generalization gradient is probably zero (8).

PRIMARY STIMULUS-INTENSITY GENERALIZATION

In a second study (5) employing the same general apparatus arrangement, Hovland attempted to determine the quantitative

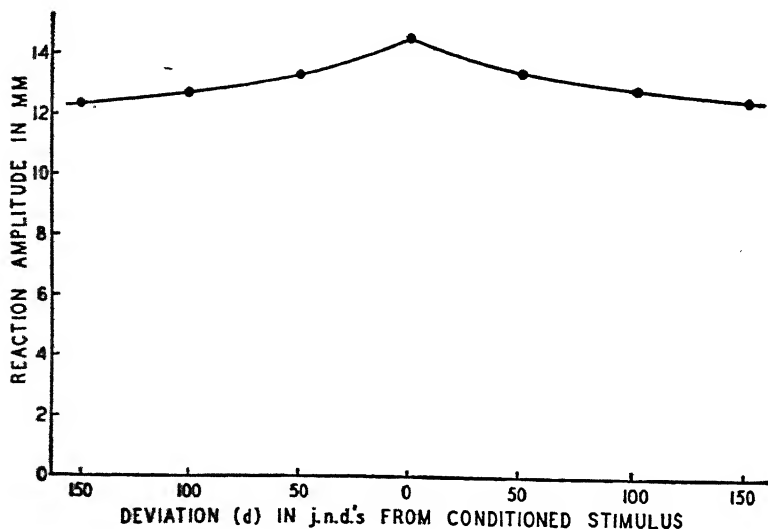


FIG. 43. Empirical stimulus-intensity generalization gradient of the conditioned galvanic skin reaction plotted from data published by Hovland (5). Note that while the gradients slope downward with increasing degrees of deviation from the point originally conditioned, the steepness of the slope is distinctly less than that of the stimulus-quality generalization shown in Figure 42.

law of stimulus-intensity generalization. The procedure was to condition a given intensity of a simple sinusoidal sound wave to the galvanic skin reaction, and then test other intensities of the same frequency at 50 j.n.d. intervals. While the results of general-

ization were seriously complicated by specific effects of intensity which were quite independent of generalization, it is believed that these latter effects were substantially eliminated by pooling the results of two groups of subjects, one in which the reaction amplitude was determined for intensities greater than that conditioned and one in which the determination was made for intensities which were less. The outcome of this experiment is shown by the circles in Figure 43. A negative growth function has been fitted to these values; this is represented by the smooth curve running among the circles. The fit is reasonably good.

It is evident from an inspection of Figure 43 that stimulus intensity also manifests a generalization gradient, but that the rate of fall of the gradient per j.n.d. of deviation from the point conditioned is distinctly less than is that for stimulus-quality generalization as shown in Figure 42. The latter has a fractional rate of decremental change per j.n.d. of deviation from the point conditioned of approximately $1/33$, whereas the former has an F-value of approximately $1/77$.

THE CONCEPT OF EFFECTIVE HABIT STRENGTH ($s\bar{H}_R$)

From the foregoing it is evident that the simple notion of habit strength, as indicating merely the strength of connection between the stimulus and the reaction involved in the original reinforcement process, must be radically expanded before the influence of learning on functional activity is to be understood and represented in a realistic manner. It is true that the various principles of reinforcement when perfected will presumably enable us to predict with precision the strength of the connection between the conditioned stimulus and the associated reaction. This is all right so far as it goes, but it represents only a small portion of the zone of reaction evocation potentialities set up by a given reinforcement. The strength of the connections at the other points of the zone can be determined only from a knowledge of the strength of the receptor-effector connection (sH_R) at the point of reinforcement and the extent of the difference (d) between the position of the conditioned stimulus (\hat{S}) and that of the evocation stimulus (S) on the stimulus continuum connecting them. Thus there emerges the concept of functional or *effective habit strength*, which we shall represent by the symbol $s\bar{H}_R$. This symbol will be used to designate the habit strength throughout the entire zone of habit forma-

tion which is set up by a given reinforcement process, or, with modifications (${}_s\bar{H}_R$, according to conditions), the summation of the effects of two or more reinforcement processes. The symbol ${}_sH_R$ will be reserved, as before, to indicate the strength of habit at the point of reinforcement; i.e., when $d = \text{zero}$,

$${}_s\bar{H}_R = {}_sH_R.$$

THE CONCEPTS OF STIMULUS DIMENSION AND AFFERENT GENERALIZATION CONTINUUM

It is clear from the now familiar causal relationship $S \longrightarrow s \longrightarrow r \longrightarrow R$ that in the evocation process (1) the stimulus energy (S) determines which receptor shall be activated and the occasion of its activation, (2) the nature of the receptor thus activated determines the detailed characteristics of the receptor discharge (s), and (3) the reaction (R) is only *indirectly* a function of S by virtue of the fact, and only to the extent, that s stands in a one-to-one relationship to S . There is reason to believe that this parallelism is never exact and that certain factors such as afferent neural interaction may produce marked deviations.

These considerations have definite implications for certain phenomena of stimulus generalization. Thus it is clear that there can be no primary stimulus generalization unless there is some parallel physical variability in the stimulus energy to serve as its basis; for example, there could be no generalization in the stimulus dimension of frequency or amplitude of sound waves if sound waves did not present such dimensions of variability. Secondly, generalization cannot take place on a given stimulus dimension if the relevant receptor does not respond differentially to variability in that dimension; for example, organisms which are color blind, i.e., those whose light receptors do not yield differential responses to variations in wave length of light, can hardly be expected to show a generalization gradient along this stimulus dimension. Accordingly there emerge the contrasted concepts of *stimulus dimension* and *afferent generalization continuum*, the latter being the differential afferent response (s) corresponding in varying degrees to variation in a given stimulus continuum. With few exceptions the receptors of normal higher organisms appear to yield afferent-generalization continua for all the physical stimulus dimensions to which they respond at all. Conversely, for every empirically observed *primary*

generalization dimension, some measurable dimension of the physical situation has usually been found.

Since most stimulus energies vary in more than one dimension, it comes about that any given bit of learning is likely to set up generalization gradients along several continua simultaneously. Inasmuch as the relevant stimulus dimensions may vary independently, the resulting mixture of several generalization gradients in a single generalization situation often greatly complicates the interpretation of experimental results involving generalization. This is particularly true in the field of vision, where there may be combined the stimulus dimension corresponding to white light and the innumerable afferent generalization continua arising from the simultaneous combinations of two or more wave lengths. To complicate matters still further there is the neural interaction (p. 42) of processes going on in different parts of the retina, which makes the afferent discharge (\bar{s}) of a given retinal element not merely a function of the stimulus energy (S) impinging on it but also of the energies S' , S'' , etc., impinging on neighboring elements at or about the same time. This complication becomes especially apparent in figured or spatially patterned stimulus situations in which there may emerge such generalization continua as degrees of curvature or angle of outline, size of figure, brightness, contrast or difference between portions of the area stimulated, the angle of rotation of the figure, and so on. When numerous physical dimensions are mixed in various ways and, particularly, where interaction occurs between different parts of the retina, the nature and amount of the generalization effects are extremely difficult to predict, as the extensive experimental investigations of Lashley have shown (10). There is reason to hope, however, that these problems will finally yield to the joint and systematic study of primary generalization gradients and gradients of afferent neural interaction. Certain investigations of the *Gestalt* psychologists should prove valuable in clarifying the latter type of problem.

IN WHAT UNITS SHALL THE DIFFERENCE BETWEEN THE CONDITIONED STIMULUS AND THE EVOCATION STIMULUS IN PRIMARY GENERALIZATION BE MEASURED?

Discrimination investigations show with considerable clarity that while s is a function of S , the relationship is usually by no means linear, and frequently it is not even monotonic. A well-known

example of the latter type of irregularity concerns the octave in auditory wave frequency. There is a tendency for generalized reactions to be evoked more strongly by stimuli which are even multiples of the frequency originally conditioned than by certain intermediate rates (9), a fact which is in conflict with the principle represented by Figure 42. The present *a priori* unpredictability in the character of many receptor responses as a function of the several stimulus dimensions is presumably due to our ignorance regarding the physiology of the receptors. Because of these irregularities it will probably be impossible to represent all generalization gradients as any uniform function of the stimulus dimensions involved.

There remains, however, the possibility of expressing generalization gradients in terms of distances on the afferent generalization continuum. The natural unit of measurement on this continuum is the discrimination threshold, or j.n.d. This is a difference between two stimuli on a given stimulus dimension (the other dimensions remaining constant) such that at the limit of discrimination training the organism will consistently give differential reactions to the two stimuli on 75 per cent of the trials. Presumably because the process of discrimination involves the joint effect of variability in the stimulus dimension and the corresponding afferent reaction of the receptor, the generalization gradients in general appear to be rather accurately and simply expressible as negative growth functions of the stimulus dimension when transmuted into j.n.d. units.

GENERALIZATION BY MEANS OF IDENTICAL STIMULUS COMPONENTS

There may now be mentioned a second form of stimulus generalization, that which arises indirectly because conditioned stimuli are not simple but are normally compounded of the simultaneous discharge of a very great number of distinct receptors. Let it be supposed, for example, that a salivary reaction has been conditioned to a compound stimulus consisting of a group of sound waves produced by an organ pipe (S_s) and a group of light waves produced by an incandescent filament (S_l), and that each of the two stimulus aggregates has independently acquired a superthreshold potentiality for evoking the reaction. Now, if a second stimulus situation consisting of the vibrations produced by the organ pipe

(S_2) were presented either alone or in combination with a cutaneous vibration (S_c), say, the reaction would be evoked, owing to the presence in the second stimulus situation of the originally conditioned auditory component (S_1). The operation of this principle has been investigated by the author at a gross molar level in connection with an experimental study of generalizing abstraction or concept formation (6); it has also been utilized extensively by Thorndike (13) in the explanation of certain forms of training transfer.

It is tempting to assume with Guthrie (2) that all primary generalization is built on this model. Involved in such an hypothesis there is the implicit assumption that the afferent discharge initiated by every stimulus energy consists of a large number of afferent "molecules," and that the contiguous receptor discharges on any given stimulus continuum have a considerable portion of their afferent molecules in common while differing with respect to certain others. Thus one receptor discharge might consist of the molecules a, b, c, d, e, f, g , whereas the adjacent one on the afferent generalization continuum would consist of the molecules b, c, d, e, f, g, h , the next one would consist of c, d, e, f, g, h, i , and so on. One discrimination threshold on such a continuum would be the physical measure of the qualitative or quantitative variation in the stimulus energy which would change enough afferent molecules so that a subject, at the limit of training, would react differentially to the two stimuli on 75 per cent of the trials. It is quite possible that something of this nature will turn out to be the ultimate physiological explanation of primary generalization. As yet, however, proof is lacking on the molecular level, and there seems no immediate prospect of securing a critical test of the hypothesis. Meanwhile we must get along as best we can with a molar analysis based on empirically determined functional relationships, e.g., those presented graphically in Figures 42 and 43.

SECONDARY STIMULUS GENERALIZATION

Except under certain special circumstances, such as those of sensitization (7, p. 431) or "long" trace conditioned reactions, conditioned stimuli probably do not show generalization into other receptor modes. Yet we may recall the name of a person with about equal probability on seeing either his face or the back of his head, at the sound of his voice or even his footstep. Such be-

havior presumably comes about not through primary stimulus generalization, but indirectly through each reaction being specifically learned. For example, a salivary reaction may be conditioned to a tactile vibration, to an auditory vibration, and to a flash of light. Since each of the three stimuli leads indifferently to the salivary reaction, this type of habit organization constitutes a learned receptor-effector convergence, quite distinct from the convergence produced by primary generalization (Figure 41).

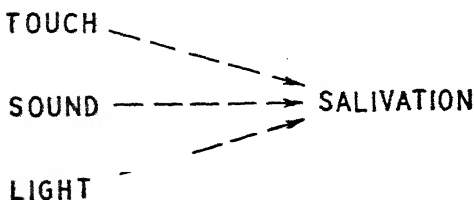


FIG. 44. Diagram of a specifically learned convergent excitatory mechanism. Each stimulus is assumed to have been conditioned to the salivary reaction on a separate occasion.

Such a learned convergence is represented diagrammatically in Figure 44.

Receptor-effector convergence is of particular importance in behavior theory, since it appears to be a medium of the automatic transfer of training effects. It is significant in the present context because, as a special case of such habit transfer, it seems to mediate what is known as secondary stimulus generalization.

An apparent case of secondary stimulus generalization has been reported by Shipley (12) and verified by Lumsdaine (3, p. 230). These investigators presented a subject with a flash of light followed by the tap of a padded hammer against the cheek below the eye, thus conditioning lid closure to the light flash. Next, the same subject was repeatedly given an electric shock on the finger. This evoked not only a sharp finger withdrawal from the electrode, but lid closure as well. Finally the flash of light was delivered alone. It was found in a considerable proportion of the subjects of both experiments that during this latter manoeuvre the light evoked finger retraction *even though the former had never been associated with either the shock or the finger retraction*. The interpretation is that the light evoked the lid closure, and the proprioceptive stimulation produced by this act (or some other less conspicuous act conditioned at the same time) evoked the finger retraction.

Lumsdaine's photographic records (3, p. 231) of the process tend to support the view that in this experiment the wink reaction served as a mediating agent, since they show that, typically, when the light evoked finger retraction the lid closure usually took place

between the flash of the light and the finger movement. Occasionally, however, the two reactions occurred at the same time, and sometimes the finger movement even preceded the blink. This, of course, could not have happened if the finger retraction was evoked by the proprioceptive stimuli arising from the lid closure. It is possible, however, that numerous other reactions were conditioned at the same time as the wink, and that proprioceptive stimuli from all of them became conditioned to finger retraction. If occasionally the lid closure should have occurred later than the other reactions, the proprioception from the latter might easily have evoked the finger retraction alone. While these considerations complicate the interpretation of Shipley's results to the extent that they do not constitute an unequivocal proof of the mechanism of secondary generalization, the experiments do demonstrate the existence of secondary generalization

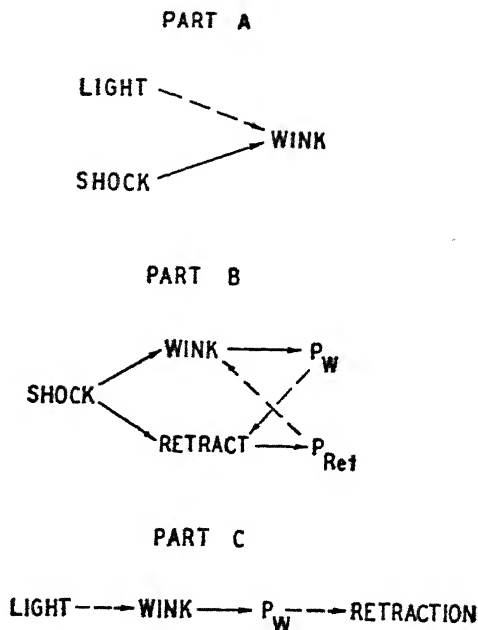


FIG. 45. Diagrammatic representation of the evolution of secondary stimulus generalization in the Shipley-Lumsdaine experiments. Part A shows the basic convergent mechanism, the light-wink portion having been set up by means of a previous conditioning process. Part B represents the conditioning of the proprioceptive stimuli of two reactions evoked by a shock, each to the other reaction through simultaneous occurrence closely associated with reinforcement, i.e., cessation of the shock. Part C shows the final indirect generalization. The light evokes the wink (from Part A); the wink produces a proprioceptive stimulation (P_W from Part B); and P_W evokes a finger retraction as conditioned in Part B. Thus the finger retraction has been indirectly generalized from the shock to the light through the mediation of the wink reaction upon which both light and shock as stimuli converge. Throughout this diagram the arrows with solid shafts represent receptor-effector connections which were in existence at the outset of the learning process here under consideration, and the arrows with broken shafts represent connections set up during the experiment.

and at the same time offer a convenient illustration of a plausible explanatory mechanism. This is shown diagrammatically in Figure 45, the legend of which gives a somewhat detailed explanation.

It is evident that when to the complexities of primary generalization mentioned earlier in the chapter there are added those naturally arising from secondary generalization (p. 191), the task of predicting generalization effects becomes almost hopeless because secondary generalization is so largely dependent on fortuitous elements in the history of the individual organism, and these are usually not known to the investigator. In this connection we may note the great facility of normal human beings in the acquisition and use of speech reactions and the recent experimental evidence that speech reactions operate in subtle ways to mediate secondary generalization (1). Because of these considerations, the results of introspective or verbal reports of the existence of generalization continua which do not conform with a reasonable approximation to some objective stimulus continuum in situations where interaction effects are presumably not marked are open to a certain amount of doubt. When uncertainty arises as to the status of such dimensions, the situation should be clarified by a comparison of the results of introspection with the generalization gradients produced in naïve organisms presumably lacking the mediating speech habits.

An important conclusion flowing from the preceding considerations is that *the common-sense notion of similarity and difference is based upon the presence or absence of primary generalization gradients, whereas so-called logical or abstract similarities and differences¹ arise from secondary, learned, or mediated similarities and differences, particularly those mediated by verbal reactions.*

THE "STIMULUS-LEARNING" AND "STIMULUS-EVOCATION" PARADOXES AND THEIR RESOLUTION

The conventional representation of learning as the formation of simple bonds gives rise to certain paradoxes. The flux of the world to which organisms must adapt has infinite variety, and therefore stimuli, especially conditioned stimuli, are never exactly repeated. But superthreshold (adaptive) reaction potentials (p. 326 ff.)

¹ Consider, for example, the similarity among weapons: this lies hardly at all in the receptors activated. Other examples in point are: similarity or difference in degree of value, weight, height, etc., as represented by numbers; as primary stimuli, 10 and 90 are hardly as different as are 10 and 12, yet $90 - 10$ is forty times as great as is $12 - 10$.

usually require more than one reinforcement to be raised above the reaction threshold. Since the stimuli are not exactly repeated, how can more than one reinforcement occur? This is the *stimulus-learning* paradox. But even if a superthreshold bond should be established, it becomes a mystery how it could ever evoke a reaction at a time of need, because the exact stimulus would probably

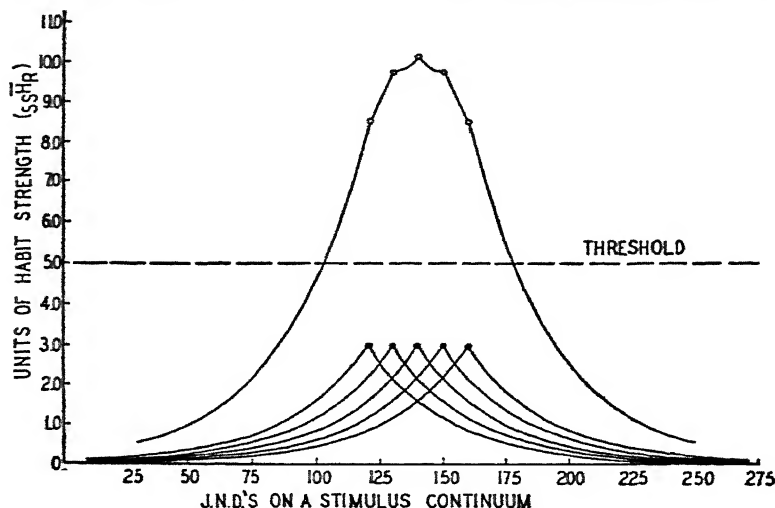


FIG. 46. Graph showing how the subthreshold primary stimulus generalization gradients from five distinct points on a stimulus continuum theoretically may summate to superthreshold values not only at the points of reinforcement but at neighboring points which have not been reinforced at all. Solid circles represent the results of single reinforcements; hollow circles represent the results of summation. The reaction threshold is arbitrarily taken at 5. Note that the major reaction tendency accumulates especially at the midpoint of the distribution of stimulated points on the continuum, but that superthreshold reaction potentialities extend beyond the range of the points conditioned.

never again be encountered. This is the *stimulus-evocation* paradox. The principle of primary stimulus generalization now available enables us to resolve both of these paradoxes.

Let it be supposed, in a particular reinforcement situation in which an effective habit strength of five habits is a minimum necessary to evoke reaction, that each reinforcement connects the conditioned stimulus to the reaction with a strength of three habits; that five reinforcements occur, the five conditioned stimuli involved falling on the same stimulus continuum at uniform intervals of

10 j.n.d.'s; that for a given potential stimulus each j.n.d. of additional deviation on the stimulus continuum from the point conditioned decreases the effective habit strength by approximately one thirty-third; and that the several habit strengths thus active at a given point on the stimulus continuum summate to produce a joint habit strength, as would the number of reinforcements necessary to produce each if they were to be given in some standard reinforcement sequence (Postulate 4).

The dynamics of this supposititious situation are represented diagrammatically in Figure 46. The habit strengths of primary reinforcement are shown by the five solid circles, the generalization gradients of each being indicated by the negative growth curves sloping downward in two directions. From an inspection of these overlapping gradients it is evident that in addition to the three habits arising from the reinforcement at a given point, there are to be combined four lesser generalization values. A little further study will show that the nearer to the middle of the distribution of conditioned stimuli a point of reinforcement stands, the larger, upon the whole, will be the generalization values to be combined. Combining these five $s\bar{H}_R$ values at each of the five points of reinforcement and at intervals of 5 j.n.d.'s on either side, there is obtained the series of summation values represented by the upper curve drawn through the five hollow circles. An inspection of the latter curve discloses the following:

I. A number of subliminal reinforcements conditioning the same reaction to distinct stimuli closely spaced along a stimulus continuum may yield an unbroken superthreshold zone of habit strengths extending well beyond the range of the conditioned stimuli in question.

II. The point of maximum habit strength tends to fall at the middle of the distribution of conditioned stimuli.

III. Points on the stimulus continuum between two points of reinforcement, but themselves not reinforced at all, have an effective habit strength only a little less than the mean of the strengths of the adjacent reinforcement points.

IV. Points on the stimulus continuum falling beyond the range of the stimuli involved in the conditioning process also rise above the reaction threshold but in progressively smaller amounts the more remote they are from the central tendency of the distribution of the stimuli conditioned.

By the first item in the above summary, isolated stimuli subliminally reinforced only a single time ultimately become supraliminal through the summation of effective habit strength ($_{ss}\overline{H}_E$) generated at the point in question with that generalized from other points of reinforcement; thus is resolved the stimulus-learning paradox. By the summation of generalized effective habit strengths from adjacent stimulus points of reinforcement, supraliminal habit strengths evolve at points which have never been reinforced at all; thus is resolved the stimulus-evocation paradox.

SUMMARY

Under favorable experimental conditions in a learning situation both the conditioned and the unconditioned stimuli may be held relatively constant. In this way a connection is said to be set up in the nervous system between the afferent discharge (\dot{s}) aroused by the conditioned stimulus (\dot{S}) and the efferent discharge (r) which leads to the reaction (R). Actually, however, very much more than this results; the reaction is conditioned not only to a tone (\dot{S}) but to a whole zone of tones of other pitches and intensities spreading in both directions along each dimension from the point conditioned. All of these stimuli are functionally equivalent in that they have the capacity to evoke the same reaction. This spreading of the results of learning to other stimuli is called primary stimulus generalization. The fact that many stimuli alike possess the potentiality of evoking the same reaction constitutes *primary stimulus equivalence*.

Experiments show, however, that the strength of the habit generalized to stimuli other than the one originally conditioned diminishes progressively as the difference between \dot{S} and S increases. When the magnitude of this difference is measured in units of the discrimination threshold (j.n.d.), the gradient of generalization closely approximates a simple negative growth or decay function.

The introduction of the phenomenon of primary stimulus generalization makes it quite clear that knowing the habit strength at the approximate points of reinforcement is now sufficient to enable us to predict the reaction potentiality, motivation (drive) remaining constant. The actual or effective habit strength mobilizable by a given evoking stimulus (S) is a joint function of the habit strength at the point or points reinforced and the difference

on their generalization continuum between the point or points of reinforcement and the stimulus point of evocation. There thus emerges the necessity for a new symbolic construct, that of effective habit strength ($s\bar{H}_R$).

The concept of "stimulus dimension" may be contrasted with that of "afferent generalization continuum." The first of these expressions refers to the physical characteristics of the stimulus energy; the second, to the characteristics of the corresponding afferent discharge initiated by the action of the stimulus energy upon the receptor. Discrimination experiments indicate that there is not a one-to-one parallelism between these two variables. It is held that the number and nature of the various primary generalization gradients are caused jointly by the nature of the stimulus energy and the nature of the receptor response. The j.n.d. is also a joint function of the nature of the stimulus energy and the nature of the receptor response. It is probably because of this that generalization is a more simple and uniform function of distance on the generalization continuum when the latter is measured in j.n.d.'s than when measured in the ordinary physical units of the stimulus.

A second form of stimulus generalization applies to stimulus compounds. The equivalence of two or more stimulus compounds in their capacity to evoke the same reaction may depend upon (1) the presence in each compound of certain identical (or similar) stimulus elements or aggregates, (2) the reaction becoming conditioned to the several stimulus elements or aggregates in one stimulus compound, and (3) the common stimulus element in the second stimulus compound tending to evoke the reaction much as it did in the original compound.

The range of primary stimulus generalization has limitations, particularly in the spread of reaction tendencies from one receptor mode to another. Stimulus equivalence in such cases is brought about by an indirect process known as secondary generalization. This evolves by a series of steps: (1) energies of distinct stimulus modes become conditioned to the same reaction by direct reinforcement; (2) a second reaction may later be conditioned to one of the stimulus energies; (3) still later, if some other stimulus also conditioned to the first reaction but *not* to the second should impinge on the organism, that stimulus will evoke the first reaction, and the proprioception of this reaction will evoke the second reaction. A stimulus-response chain of this kind mediates secondary

or indirect stimulus generalization, a second form of stimulus equivalence.

The summation of overlapping primary stimulus generalizations, even if definitely subliminal at their point of reinforcement and even if each stimulus point is reinforced only once, may be shown to raise the effective habit strength above the reaction threshold not only at the points reinforced but at neighboring stimuli which have never been reinforced at all. In this way are explained both the paradox of the occurrence of superthreshold learning where the conditioned stimulus is never exactly repeated, and the paradox of reaction evocation where the evoking stimulus has never been associated with the reaction evoked.

In view of the preceding considerations we may formulate Postulate 5:

POSTULATE 5

The effective habit strength $s\bar{H}_R$ is jointly (1) a negative growth function of the strength of the habit at the point of reinforcement (\bar{S}) and (2) of the magnitude of the difference (d) on the continuum of that stimulus between the afferent impulses of \bar{s} and s in units of discrimination thresholds (j.n.d.'s); where d represents a qualitative difference, the slope of the gradient of the negative growth function is steeper than where it represents a quantitative difference.

From Postulates 4 and 5 there follows an important corollary; because of the frequency of its use it is here given special prominence:

MAJOR COROLLARY I

All effective habit tendencies to a given reaction, whether positive or negative, which are active at a given time summate according to the positive growth principle exactly as would the reinforcements which would be required to produce each.

NOTES

Mathematical Statement of Postulate 5

This postulate is expressed concisely by the equation:

$$s\bar{H}_R = sH_R e^{-j'd} \quad (29)$$

where,

sH_R is as given in equation 16,

d is the difference between S and \bar{S} in j.n.d.'s,

and

j' is an empirical constant of the order of .01 in the case where d is a qualitative difference but of the order of .006 where d is a quantitative difference.

Mathematical Statement of Major Corollary I

$$ss\bar{H}_R = \Sigma_1 - \frac{\Sigma_2}{M} + \frac{\Sigma_3}{M^2} - \dots + (-1)^{n-1} \frac{\Sigma_n}{M^{n-1}}, \quad (30)$$

where Σ_1 is the simple sum of the items to be combined, Σ_2 is the sum of the products of all combinations taken two at a time, Σ_3 is the sum of all products taken three at a time, etc., and M is the physiological limit of the learning process, in this case assumed to be 100.

The Hovland Stimulus Generalization Gradients

Hovland's numerical values from which Figure 42 was plotted are as follows:

<i>No. j.n.d.'s distant from point of reinforcement (d)</i>	<i>Amplitude of galvanic skin reaction in millimeters (A)</i>
0	18.3
25	14.91
50	13.62
75	12.89

The negative growth function fitting these data rather well is:

$$A = 18.3 - 6(1 - 10^{-.0135d}),$$

where A is the amplitude (in millimeters) of the galvanic skin reaction to stimulation, 12.3 represents the asymptote or limit of fall of the value of A , 6 is the maximum amount of change in A due to generalization, and .0135 is a constant depending in part on the steepness of slope of the generalization function and in part on the units employed. This equation is represented by the smooth curves drawn among the data points in Figure 42.

The smooth curve drawn through the data points of Figure 43 corresponds to the equation:

$$A = 14.3 - 2.24(1 - 10^{-.0061d}).$$

The Method of Deriving from Empirical Values the Constants for the Growth Function Fitted to Hovland's Data

It was concluded from an inspection of the data in graphic form that the function probably was a decay or negative growth variety with an asymptote considerably greater than zero, i.e., that the equation would probably be of the form:

$$A = a + (b - a)10^{-kd}.$$

In this equation, b is the value of A when $d = 0$. This is given directly by the data table as 18.3. Substituting, the equation becomes:

$$A = a + 18.3 - a$$

In this equation, A and d are given by the table of empirical values, which leaves two unknowns, b and a . These values are found by means of simultaneous equations, three pairs of which may be set up from Hovland's empirical results.

* This equation was derived by Arthur S. Day.

Taking $d = 50$ for one equation, and $d = 75$ for the other, and substituting the corresponding A -values from Hovland's data, we have:

$$13.62 = a - \frac{18.3 - a}{10^{50h}}$$

$$12.89 = a - \frac{18.3 - a}{10^{75h}}$$

Solving this pair of simultaneous equations we find that $a = 11.1$ and $h = .008$.

Setting up the two remaining significant simultaneous combinations afforded by Hovland's data we obtain additional values for both a and h . The whole series of values is as follows:

d	a	h
25 and 50	12.9	.017
25 " 75	12.5	.015
50 " 75	11.1	.008

Taking the approximate central tendency of the three values for each constant we have, $a = 12.3$ and $h = .0135$. Substituting, we have:

$$\begin{aligned} A &= 12.3 + \frac{18.3 - 12.3}{10^{.0135 d}} \\ &= 12.3 + 6 \times 10^{-.0135 d}, \end{aligned}$$

which is the equation sought.

The Derivation of Figure 46

At the outset it is assumed that single subliminal reinforcements were made at five points on an afferent generalization continuum: 120, 130, 140, 150, and 160 j.n.d.'s distant from a rather remote common point marked zero in the figure.

Next, the generalization value from each point of reinforcement was calculated by the equation:

$${}_s\bar{H}_R = 3 \times 10^{-.0135 d}. \quad (31)$$

For reasons given in the text (p. 186), this equation has been adapted from that derived from Hovland's data as a sounder and more general equation for the generalization gradient. In this way were obtained the results given in the following table:

<i>ce in j.n.d. (d)</i>	<i>Generalized effective habit strength (${}_s\bar{H}_R$) in habs</i>
0	3.0
5	2.57
10	2.20
15	1.88
20	1.61
30	1.18
40	.87
50	.63
60	.46
70	.34
80	.25
90	.18
100	.13
110	.10

From this table were obtained the generalization values sloping downward and away in each direction from the crests which appear in the lower part of the figure at the five points of reinforcement.

The next step in the process was to combine the overlapping generalization tendencies which appear at every point on the stimulus continuum as five items. For example, at 120 the first item is 3.0 because here $d = \text{zero}$. Next, there is the generalization value of the gradient originating at 130, which is 10 j.n.d.'s distant. By the above table this would yield 2.20 habs. Then there is the generalization value originating at 140. Since 140 is 20 j.n.d.'s from 120, $d = 20$, which, by the above table, corresponds to a generalization value of 1.61 habs. In a similar way the other two $s\bar{H}_R$ values with d 's of 30 and 40 are shown by the table to be 1.18 and .87, respectively.

We thus have the problem of combining 3.0, 2.2, 1.61, 1.18, and .87 habs. How shall this be done? The hypothesis which fits in best with various related empirical observations is that the generalized effects of learning summate in the same way as do the effects of repetitions in the learning process. The principles according to which repetitions of reinforcement combine to produce habit tendencies have been explained in considerable detail in Chapter VIII and are stated concisely in Postulate 4 and in equations 1, 26, and 30. It will be sufficient here only to say that each repetition was supposed to increase the amount of habit strength by a constant factor (e.g., 1/10) of the learning potentiality not yet realized in actuality. This means that the amount of habit strength contributed by one repetition of reinforcement late in the learning process is very much less than that contributed by one repetition at the beginning. In a similar manner, a block of five repetitions given late in the learning process will contribute less to the habit strength than an exactly similar block of five repetitions early in the process. It was assumed that the five generalized tendencies summate just as would the number of repetitions required to produce each. Thus one might substitute the value of $s\bar{H}_R$ in equation 26:

$$sH_R = m(1 - 10^{-iN})$$

and solve for N in the case of each of the five values listed above, add together the set of N 's thereby obtained, and, finally, substitute the sum of the five N 's in the same equation, this time solving for sH_R ; the result of the last operation would constitute the summation required.

The above procedure, while conceptually simple, is very clumsy mathematically. Accordingly the mathematical implications of the assumption have been worked out for the general case where n values must be summated. This takes the form of the equation given in the second terminal note above (30). The meaning of that equation will be made clear by the following example:

$$Z_1 = 3.00 + 2.20 + 1.61 + 1.18 + .86 = 8.85.$$

$$\begin{aligned} Z_2 &= 3.00 \times 2.20 + 3.00 \times 1.61 + 3.00 \times 1.18 + 3.00 \times .86 + 2.20 \times 1.61 \\ &\quad + 2.20 \times 1.18 + 2.20 \times .86 + 1.61 \times 1.18 + 1.61 \times .86 + 1.18 \times .86 \\ &= 6.60 + 4.83 + 3.54 + 2.58 + 3.54 + 2.60 + 1.89 + 1.90 + 1.38 + 1.01 \\ &= 29.87 \end{aligned}$$

$$\begin{aligned} Z_3 &= 3.00 \times 2.20 \times 1.61 + 3.00 \times 2.20 \times 1.18 + 3.00 \times 2.20 \times .86 + 3.00 \\ &\quad \times 1.61 \times 1.18 + 3.00 \times 1.61 \times .86 + 3.00 \times 1.18 \times .86 + 2.20 \times 1.61 \\ &\quad \times 1.18 + 2.20 \times 1.61 \times .86 + 2.20 \times 1.18 \times .86 + 1.61 \times 1.18 \times .86 \\ &= 10.63 + 7.79 + 5.68 + 5.70 + 4.15 + 3.04 + 4.18 + 3.04 + 2.24 + 1.63 \\ &= 48.08 \end{aligned}$$

$$\begin{aligned}\Sigma_4 &= 3.00 \times 2.20 \times 1.61 \times 1.18 + 3.00 \times 2.20 \times 1.61 \times .86 + 3.00 \times 2.20 \\ &\quad \times 1.18 \times .86 + 3.00 \times 1.61 \times 1.18 \times .86 + 2.20 \times 1.61 \times 1.18 \times .86 \\ &= 12.54 + 9.14 + 6.70 + 4.90 + 3.59 \\ &= 36.87\end{aligned}$$

$$\begin{aligned}\Sigma_5 &= 3.00 \times 2.20 \times 1.61 \times 1.18 \times .86 \\ &= 10.78.\end{aligned}$$

$$\begin{aligned}ss\bar{H}_R &= 8.85 - \frac{29.87}{100} + \frac{48.08}{10,000} - \frac{36.87}{1,000,000} + \frac{10.78}{100,000,000} \\ &= 8.85 - .30 + .0048 - .000037 + .00000011 \\ &= 8.55\end{aligned}$$

which is the value of $ss\bar{H}_R$ at 120 on the upper or summation graph in Figure 46. All of the other points in the summation curve were computed in an analogous manner.

In general this method of summation yields a value appreciably less than would be obtained by the simple addition of the items summated, the shrinkage being greater the nearer the individual items approach the magnitude of the physiological limit (M), in this case, 100. Because of the relatively small size of the items, the shrinkage here is slight.

REFERENCES

1. BIRGE, J. S. The role of verbal responses in transfer. Ph.D. thesis, Yale University, 1941.
2. GUTHRIE, E. R. Conditioning as a principle of learning. *Psychol. Rev.*, 1930, *37*, 412-428.
3. HILGARD, E. R., and MARQUIS, D. G. *Conditioning and learning*. New York: D. Appleton-Century Co., Inc., 1940.
4. HOVLAND, C. I. The generalization of conditioned responses: I. The sensory generalization of conditioned responses with varying frequencies of tone. *J. Gen. Psychol.*, 1937, *17*, 125-148.
5. HOVLAND, C. I. The generalization of conditioned responses: II. The sensory generalization of conditioned responses with varying intensities of tone. *J. Genet. Psychol.*, 1937, *51*, 279-291.
6. HULL, C. L. Quantitative aspects of the evolution of concepts. *Psychol. Monogr.*, 1920, *28*, No. 123.
7. HULL, C. L. Learning: II. The factor of the conditioned reflex. Chapter 9 in *A handbook of general experimental psychology*, C. Murchison, editor. Worcester, Mass.: Clark Univ. Press, 1934.
8. HULL, C. L. The problem of stimulus equivalence in behavior theory. *Psychol. Rev.*, 1939, *46*, 9-30.
9. HUMPHREYS, L. G. Generalization as a function of method of reinforcement. *J. Exp. Psychol.*, 1939, *25*, 361-372.
10. LASHLEY, K. S. The mechanism of vision: XV. Preliminary studies of the rat's capacity for detail vision. *J. Gen. Psychol.*, 1938, *18*, 123-193.
11. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
12. SHIPLEY, W. C. Indirect conditioning. *J. Gen. Psychol.*, 1935, *12*, 337-357.
13. THORNDIKE, E. L. *Educational psychology*. II. *The psychology of learning*. New York: Teachers College, Columbia Univ., 1913.

CHAPTER XIII

Some Functional Dynamics of Compound Conditioned Stimuli

Most accounts of conditioning experiments tend enormously to minimize the actual complexity of the factors involved. Indeed, this is almost a necessity; if the reports of such experiments should contain a really complete description of the process the reader would be so swamped in detail that he might easily fail to understand the main point of the experiment. The same expository difficulty is encountered by behavior theorists in perhaps an even more aggravated form and has led to the same type of misrepresentation. An example of this is our own use of the symbol sH_R . There is small doubt that such expository over-simplifications in the accounts of learning and other behavior situations have genuinely misled many persons beginning the study of behavior and, possibly, in some cases even the investigators themselves; they certainly have produced much misapprehension among the philosophical critics of behavior theory, who as a rule have little or no first-hand knowledge of the phenomena concerned and so are especially prone to such misunderstandings. In Chapter XII something was done to remedy this wholly natural yet regrettable situation by considerably expanding the concept of habit with respect to the stimulus; a parallel expansion on the response side will be presented in Chapter XVII. In the present chapter we shall seek to clarify the concept of the stimulus (S) still further by deriving a number of elementary corollaries from the conditions under which learning occurs when considered in conjunction with certain primary principles; the latter are for the most part already familiar to the reader.

THE COMPLEXITY OF THE "STIMULUS" OF A TYPICAL CONDITIONING SITUATION

In order to clarify to some degree the actual complexity of the "stimulus" involved in typical habit formation, let us consider in a little detail this aspect of what is usually regarded as one of the more simple learning situations, that of the Pavlovian conditioned

reflex (6). Previous to the beginning of the conditioning process the dog has had no food for 24 hours. It stands upon a table in the laboratory, being kept in place by loose bands attached to a wooden framework. Some weeks previous to the experiment one of its salivary ducts has been surgically diverted so that saliva is discharged through a fistula in the side of the animal's face. When the experiment actually begins, a capsule is cemented over the fistula in such a way that it collects the saliva seeping through the opening; the pressure in the capsule resulting from the entrance of the saliva is transmitted by a rubber tube to a sensitive registering device.

An electric buzzer is sounded near the dog for five seconds; two seconds after the termination of the buzzer action a small quantity of meat powder is blown into the animal's mouth by means of a rubber tube held in place by a kind of muzzle. This powder is eaten, with a profuse accompanying flow of saliva. After a few repetitions of this sequence, the dog begins secreting saliva during the seven-second interval between the beginning of the sound and the delivery of the meat powder, showing that the conditioned reflex has been set up.

The above summary description of the conditioned reflex experiment is a fairly typical example of the accounts usually given; the only conditioned stimulus element specifically mentioned as active in the situation is the buzzer. As a matter of fact, the buzzer vibration makes up only a small part of the total number of stimulus components involved. Moreover, the wave pattern of the buzzer itself, as revealed by the cathode ray oscillograph, is an exceedingly complex phenomenon and doubtless stimulates simultaneously a very large number of the ultimate auditory receptors in the cochlea.

Among the many additional components of the conditioned stimulus (*S*) not ordinarily mentioned are: the fact that the animal's two ears receive the buzzer vibrations with different intensity or in different phase, depending on (1) the direction of the bell from the dog's head and (2) the orientation of the head at the moment; the pressure of the dog's feet against the table top upon which it stands; the pressure of each of the three or four restraining bands upon the skin receptors of the dog's neck, thighs, etc.; the biting of a number of insects which may be hidden in the dog's hair; the contact of the capsule over the fistula; the pressure of the muzzle against the dog's head; the pressure of the rubber

tube in the dog's mouth; the odor of the rubber from which the tube is made, together with a large number of miscellaneous odors to which the human olfactory receptors may or may not respond; the multitude of visual stimuli of light, shade, spatial combinations, etc., arising from the laboratory lamps and reflected from millions of points within the dog's visual field; the proprioceptive stimulation arising from the external and internal muscles of the dog's eyes as they fixate one object after another about the laboratory; the infinite number and variety of proprioceptive impulses originating in the several parts of the other muscles of the animal's body as they are employed in the maintenance of the postures taken from moment to moment; the too-little understood stimulations associated with the bodily state resulting from food, water, and sexual privation, rectum and bladder pressure, etc.; and, finally, the perseverative traces of all the multitude of stimuli recently acting, whether the stimulus energy is continuing to act at the moment or not. *The conditioned stimulus in the experiment under consideration includes all of the immensely complicated stimulus elements here enumerated and many more besides;* nevertheless this list, incomplete as it is, should aid the reader somewhat in overcoming the misleading suggestion of singularity and simplicity otherwise likely to be conveyed by the S of the symbol, $_sH_R$.

THE DISTRIBUTION OF HABIT STRENGTH ACQUIRED BY THE SEVERAL COMPONENTS OF A STIMULUS COMPOUND

The law of primary reinforcement as formulated in Chapter VI presented, in the interest of introductory expository clarity, the ultra-simple view of the conditioned stimulus which we have just been at some pains to rectify. We must now consider the operation of this principle under the present expanded conception, particularly as it applies to the several types of components which may be found in a stimulus compound.

According to the "law of reinforcement" laid down earlier (p. 80), every one of the receptor discharges and receptor-discharge perseverations active at the time that the to-be-conditioned reaction occurs must acquire an increment of habit strength ($\Delta_s H_R$). The enunciation of this principle, coupled with the recognition of the multiplicity and variety of these afferent elements, at once raises numerous critical questions, one of which is: Are these incre-

ments of habit strength all of the same magnitude? The answer to this question is quite definitely that the increments of habit strength acquired by the several afferent discharges arising from the various stimulus aggregates represented by such words as "buzzer sound," "odor of food," "sight of food cup," "pressure of restraining bands," etc., differ widely. In this respect the situation is believed to be substantially as represented in Figure 47, where the thickness of the broken lines connecting the several stimulus aggregates (S 's) represents the varying magnitudes of the increments of habit strength acquired by them at a given reinforcement. The S 's shown in the diagram are, of course, far too few to more than suggest the number of actual stimulus elements, or even the number of the aggregates of stimulus elements,¹ in the typical conditioning situation.

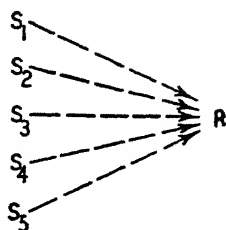


FIG. 47. This figure represents the sheaf of habit tendencies ($\Delta_s H_E$) presumably set up by each reinforcement. The thickness of the dashes leading to the several arrow points is intended roughly to represent the differences in the magnitude of the habit increments or habit loadings connecting the several stimulus elements in the conditioning situation, with the reaction process.

Recognition of the variability in the increments of habit strength acquired by the several stimulus components of a conditioning situation at once raises the question of the principles according to which the differential magnitudes of habit-strength loadings arise. This question does not permit a very definite answer, though a certain amount of experimental effort has been directed to this end. One bit of evidence comes from the Kappauf-Schlosburg experiment described above (p. 166), which showed that stimuli which have continued to act on a receptor without change for some time have a greatly diminished capacity for acquiring habit loadings. Partly for this reason it is probable that static, i.e., unchanging, elements or aggregates in a conditioned stimulus situation are considerably less potent in the acquisition of habit-strength loadings than are the more dynamic, i.e., changing, elements or aggregates. This probably is why investigators so frequently neglect to take into consideration the static or constant

¹ A *stimulus element* is defined as the action of a stimulus energy upon a single receptor organ, such as a single rod of the retina or a single touch organ of the skin. A *stimulus aggregate* is a group of stimuli which ordinarily begin and end concurrently and, in general, combine to perform the same adaptive functions.

elements of conditioned stimulus situations, sometimes with unfortunate results.

A second factor which may be of some importance in determining the habit-strength loading acquired by a stimulus aggregate in a learning situation is the intensity of the stimulus energy. Pavlov reports (6, p. 142), on the basis of admittedly inadequate empirical data, that when two stimulus energies of different intensities operate on the same receptor simultaneously, e.g., two different tones, the stronger stimulus receives a greater increment of habit strength.

A third factor is the receptor or "analyzer," which receives the stimulus energy. Pavlov reports (6, p. 143), again without adequate supporting evidence, that, other things equal, tactual stimuli seem to acquire stronger habit loadings than do thermal stimuli and that auditory stimuli are stronger in this respect than visual stimuli. Recently Zener has reported orally a well-controlled study which fully establishes this proposition for relatively low stimulus intensities.

A fourth possible factor in determining the relative habit loadings of the several components of a stimulus compound concerns whether or not a stimulus appears in a large number of conditioning situations which require a wide variety of reactions to bring about need reduction, and also in many situations requiring no reaction whatever. For example, daylight is present as a visual stimulus component in thousands of different reaction situations. It seems likely that the great number of reactions to which this stimulus component is conditioned early in life, coupled with incidental extinction effects which necessarily result from such a state of affairs, would soon largely blur out the capacity of such stimuli to be conditioned to any reaction in particular.

A fifth factor which follows as a kind of corollary to the factor of intensity is that a stimulus component which has previously been conditioned to a reaction involving strong autonomic or "emotional" aspects, e.g., a fear reaction, will presumably acquire in this indirect way a stronger habit loading than would a component not so conditioned. This would be expected on the assumption that the proprioceptive and other receptor discharges entailed by the occurrence of the conditioned reaction in question would constitute a relatively intense stimulus which, as such, would acquire a correspondingly heavy habit loading from the reinforcement process. A very weak stimulus through lack of vigorous

competition or through especially powerful reinforcement in an earlier conditioning situation might thus acquire control of a reaction yielding a powerful stimulus and in this indirect way attain the appearance, in subsequent conditioning situations, of having itself a strong capacity for acquiring habit loadings. It seems likely that this mechanism explains to a considerable extent the rôle in the learning process of what is reported introspectively as "attention."

Unfortunately as yet very little experimental effort has been directed to the solution of these problems. For this reason most of the suggestions listed above must be regarded as hardly more than conjectures suitable as points of departure for future investigations.

THE JOINT REACTION-EVOCATION POWER OF COMPOUND STIMULUS AGGREGATES

Among the problems precipitated by the highly compound character of the conditioned stimulus there is the question of the relative action-evoking power (and, presumably, of effective habit strength) of a stimulus element or aggregate (*S*) when acting in a stimulus compound as contrasted with that when it is acting alone. There are two cases; one is that in which the stimulus components are separately conditioned to the same reaction and are later tested for power of reaction evocation by being presented to the subject simultaneously as a stimulus compound. The other is that in which the stimulus components are presented simultaneously as a compound during the conditioning process and are later tested for power of reaction evocation by being presented to the subject separately. Here, as in so many other aspects of behavior science, really adequate empirical investigations are largely lacking. However, some experimental results are available which are useful for illustrating the nature of the problems and for suggesting plausible hypotheses, even if not adequate to serve as a basis for final decisions.

We proceed now to the consideration of the empirical evidence concerning the case where the stimulus components are conditioned separately to the same reaction. In one illustrative experiment eight human subjects were first conditioned to a weak light, the unconditioned stimulus being a brief electric shock and the response recorded, the galvanic skin reaction (2). Next, a weak vibratory

stimulus applied to the forearm was paired with the shock and thus conditioned to the galvanic skin reaction. When presented alone the light (S_l) evoked a mean reaction (R_l) of 3.5 millimeters, and the vibrator (S_v) evoked a mean reaction (R_v) of 3.6 millimeters. When both stimuli were presented simultaneously (S_{l+v}), they jointly evoked a mean reaction (R_{l+v}) of 4.4 millimeters.

Since the two stimulus components were of almost equal reaction-evocation strength when presented separately, it is reasonable to suppose that they contributed about equally to the joint evocation when they were presented simultaneously, i.e., that each contributed approximately half of the 4.4 millimeters of the joint evocation, or 2.2 millimeters. These results, which are typical of a number of fairly comparable sets now available, indicate a definite shrinkage in the reaction-evocation power (and so, presumably, of effective habit strength) at the command of stimulus components in combination, as compared with that when acting separately.

A simple quantitative index of this shrinkage is obtained by dividing the amplitude of the reaction evoked by the joint stimulation by the sum of the amplitudes evoked by the separate stimulations:

$$\frac{R_{l+v}}{R_l + R_v} = \frac{4.4}{3.5 + 3.6} = \frac{4.4}{7.1} = .62-$$

This is interpreted to mean that a stimulus when presented jointly with another evokes only about 62 per cent as great a reaction as it does when presented separately, thus showing a shrinkage of 38 per cent, or about a third. A comparable experiment (2), also employing eight human subjects, yielded the following values:

$$\frac{R_{l+v}}{R_l + R_v} = \frac{3.91}{2.2 + 3.7} = \frac{3.91}{5.9} = .66 +.$$

This experiment also shows a shrinkage of almost exactly one-third.

The second case of the reaction-evocation power of compound stimuli is the reverse of that just considered. This is illustrated by a simple permutation of the series of experiments outlined above, the present experiment likewise employing eight human subjects (2). The light and the vibrator, presented simultaneously, were paired with the shock. The mean amplitudes of the conditioned galvanic skin reaction evocable by the compound and by the separate components were then determined. It was found that the light alone (S_l) evoked a mean reaction amplitude of 2.5 milli-

meters; the vibrator alone (S_r) evoked a mean reaction amplitude of 2.9 millimeters; and the two components presented as a compound (S_{i+r}) evoked a mean reaction amplitude of 3.3 millimeters. Despite the difference in the arrangement of this experiment, the shrinkage of the reaction-evocation power of the stimulus components when in a compound as compared with that when presented separately may be calculated by the same formula:

$$\frac{R_{i+r}}{R_i + R_r} = \frac{3.3}{2.5 + 2.9} = \frac{3.3}{5.4} = .61 +.$$

The calculation yields a value within the range of experimental error of that obtained from the experiments falling under case one.

Another exactly comparable experiment from the same study, also employing eight human subjects, yielded the following result:

$$\frac{R_{i+r}}{R_i + R_r} = \frac{2.8}{1.4 + 2.8} = \frac{2.8}{4.2} = .66 +.$$

To the two cases of compound stimulation already considered there may now be added a third case which is of special interest because it is neutral so far as possible interaction effects arising from the circumstances surrounding the conditioning are concerned. This is illustrated by an experiment employing eight human subjects (2) in which the $S \rightarrow R$ connection was the result of what is called *sensitization*, i.e., it was set up by merely administering the shock without the latter being paired with either the light or the vibrator. This experiment yielded the following result:

$$\frac{R_{i+r}}{R_i + R_r} = \frac{3.2}{2.2 + 2.9} = \frac{3.2}{5.1} = .63 -,$$

which is in close agreement with the results of the other four experiments.

Thus, from the point of view of the quantitative index, $\frac{R_{i+r}}{R_i + R_r}$, all three cases, so far as the available evidence goes, agree in showing that where two stimulus aggregates are concerned a shrinkage of about one-third in reaction-evocation power occurs; i.e., from this particular point of view no marked difference between the three cases appears. This may very well be the situation which will finally be revealed by further experiment.

There are, however, certain indications reported by Pavlov (6, p. 141) to the effect that where two dynamic stimulus components

are jointly conditioned to a reaction, the stronger of the two may completely dominate, obscure, or "overshadow" the weaker component in that when the latter is presented separately it will evoke no reaction whatever, though in certain indirect ways it may be shown to possess some kind of functional connection with the reaction. In this context it will be recalled that in the fourth example given above, which involved a reaction conditioned to a compound stimulus, the mean amplitude of reaction evoked by the compound (2.8 millimeters) was exactly the same as that evoked by the stronger of the two stimulus components, the cutaneous vibrator; the addition of the light to the combination seems to have added nothing to the mean amplitude of reaction. This outcome might quite possibly have been due to experimental "error," i.e., to the fact that the sample of data collected was too small to yield a sufficiently precise indication of the relative influence of the several factors involved.

After weighing the various bits of experimental evidence available, the most plausible empirical generalization concerning the dynamics of compounds of conditioned stimuli in the reaction evocation is that *stimulus aggregates conditioned to the same reaction possess, irrespective of whether the stimulus aggregates were conditioned to the reaction as separate entities or as a stimulus compound, (1) a smaller power of reaction evocation when presented jointly than when presented separately, but (2) a larger joint power of reaction evocation than does any single component when the latter is presented separately.*

THE PRINCIPLES OF HABIT SUMMATION AND OF MONOTONIC HABIT-REACTION RELATIONSHIP AS APPLIED TO STIMULUS COMPOUNDS

With some examples of the coarse empirical reaction-evocation dynamics of stimulus compounds before us, the question arises as to whether the above empirical generalizations represent primary principles or secondary principles derivable from a combination of primary principles already in the system. The answer to this type of question depends on whether or not the principle can be derived from these other and supposedly more elementary principles; if so, it is a secondary principle; if not, it may be a primary principle. It should be said at once that by this test, the dynamics of reaction generalization are second-order, or derived, principles. The major

portion of these phenomena appear to be mainly dependent upon, i.e., derivable from, two principles already more or less familiar.

The first of the primary principles in question concerns the manner in which two or more homogeneous habits, i.e., those involving the same reaction, summate to produce a joint habit strength. We encountered a situation of this kind above (p. 195) when we considered the summation of habit strengths arising from the overlapping of stimulus generalization gradients both of which were conditioned to the same reaction. In that case we had the physiological summation of habit tendencies generated by the conditioning of the same reaction to two distinct stimuli but, through the process of generalization, brought to bear on the evocation of the reaction by the impact of a single stimulus aggregate. The present situation presumably has the same dynamic state of affairs brought about by the simultaneous action of two stimulus aggregates each of which has a tendency independently to evoke the same reaction. As in the case of overlapping stimulus generalizations (Chapter XII, p. 199), the quantitative principle of summation is given by Major Corollary I. As applied specifically to the present situation, this may be restated as follows: *If two or more stimulus aggregates, each independently conditioned to the same reaction, impinge simultaneously on the receptors of the organism in question, the effective habit strengths borne by the several stimulus aggregates summate to produce a joint habit strength as would the separate effects of the number of reinforcements necessary to produce each, if such reinforcements were to be given consecutively in some standard reinforcement sequence.*

Thus, suppose that one stimulus aggregate, such as a weak light, has a habit strength to the evocation of a given reaction of 34.39 habs, and a second stimulus aggregate, such as a cutaneous vibrator, has by independent reinforcement a habit strength of 46.86 habs to the evocation of the same reaction. Now, by Table 1 (Chapter VIII, p. 115), 34.39 habs would (under certain assumed conditions) be produced by four reinforcements, and 46.86 habs would be produced by six reinforcements. On the above principle it follows that the physiological summation of the two habit strengths would yield a joint habit strength equal to that which would be produced by $4 + 6 = 10$ reinforcements, which, by Table 1, equals 65.13 habs.

The second principle presumably operating here is that of the *monotonic habit-reaction relationship* (p. 326 ff.). This is that

the strength of a reaction tendency is an increasing linear function of the effective habit strength mediating it; i.e., if

$$s_1 H_R > s_2 H_R,$$

then

$$R_{s_1} > R_{s_2}.$$

SOME COROLLARIES OF THE PRINCIPLES OF HABIT-STRENGTH
COMBINATION AND OF THE MONOTONIC HABIT-REACTION
RELATIONSHIP APPLIED TO CONDITIONED
STIMULUS COMPOUNDS

It follows from the above principles that the sum of the reaction amplitude mediated by 34.39 habs and of that mediated by 46.86 habs will be an increasing function of $34.39 + 46.86$, or 81.25, whereas the magnitude of the reaction mediated by the joint action of the two stimuli will be a corresponding function of 65.13. However,

$$65.13 < 81.25.$$

We accordingly formulate Corollary I as follows:

1. *The amplitude¹ of the reaction evoked by two stimulus aggregates acting jointly will be less than will be the sum of the reaction magnitudes evoked by the respective stimulus aggregates acting separately.* Stated in a formal manner, Corollary I becomes the following inequality:

$$R_{1+2} < R_1 + R_2.$$

Returning, now, to our empirical data we find ample illustration in all five experiments. For example, in the first experiment we find,

$$R_{1+2} = 4.4$$

and

$$R_1 + R_2 = 3.5 + 3.6 = 7.1$$

i.e.,

$$4.4 < 7.1,$$

which fully satisfies the above inequality.

A secondary corollary which flows from the same assumptions concerns the reaction magnitude mediated by the summation of

¹ This statement concerning amplitude appears to hold only for certain reactions such as the galvanic skin reaction, here used as illustrative material, the salivary reaction, and the lid reaction. For certain other types of reaction such as are normally produced by the striated muscles, probability of reaction evocation (*p*) at stimulation, latency or resistance to experimental extinction will need to be substituted for amplitude.

two habits as contrasted with that mediated by either habit alone. We have seen that the joint strength of a habit of 34.39 habs and one of 46.86 habs amounts, theoretically, to 65.13 habs, which is greater than either habit strength taken alone, i.e.,

$$34.39 < 46.86 < 65.13.$$

Therefore,

$$R_{S_1} < R_{S_1 + S_2}.$$

Generalizing from the above we arrive at our second corollary:

II. *The magnitude of the reaction tendency evoked by any one of a number of stimulus aggregates conditioned to the same reaction will be less than that evoked by two or more of them acting simultaneously.* Empirical illustration of this corollary is seen in four of the five experiments cited above. Thus 4.4 is greater than either 3.5 or 3.6, and 3.9 is greater than either 2.2 or 3.7. The slightly discordant results of the fourth experiment are probably due to limitations in the size of the sample.

Thirdly, suppose that three equally potent stimulus aggregates possess a joint strength of 71.76 habs. Now, 71.76 habs corresponds (Table 1) to twelve standard reinforcements. Thus the three stimulus components must have independent strengths equivalent to one-third of 12, or four standard reinforcements, each of which, by Table 1, corresponds to 34.39 habs. According to the principle of habit strength summation, two of these, if taken together, would summate to a habit strength equivalent to eight standard reinforcements, which (Table 1) would yield 56.95 habs. But,

$$34.39 < 56.95 < 71.76$$

i.e.,

$$s_1 H_R < s_1 H_R + s_2 H_R < s_1 H_R + s_2 H_R + s_3 H_R.$$

It follows from this and the principle of the monotonic habit-reaction relationship that,

$$R_{S_1} < R_{S_1 + S_2} < R_{S_1 + S_2 + S_3}.$$

Generalizing, we formulate our third corollary:

III. *If a number of stimulus aggregates, all equally conditioned to the same reaction, impinge upon the organism simultaneously, the larger the number of such stimulus aggregates active on a given occasion, the greater will be the amplitude of the evoked reaction.*

THE PRINCIPLES OF AFFERENT INTERACTION AND PRIMARY STIMULUS GENERALIZATION

The third principle upon which the dynamics of reaction evocation by stimulus compounds depends is that of *afferent neural interaction* (Postulate 2, p. 47). In the present context that principle may be stated as follows (3, p. 77): *Concurrent afferent impulses (s) arising from the impact of distinct stimulus energies (S) on receptors are appreciably modified by each other before they reach that portion of the central nervous system where they initiate the efferent impulses (r) which ultimately evoke reactions (R).*

The relevance of the afferent interaction hypothesis to the dynamics of stimulus compounds arises from the fact that two stimulus aggregates may, as we have already seen, act either independently of each other or in combination. But, by the principle of afferent interaction, the afferent impulses which originate in a given stimulus aggregate are somewhat different on reaching the more central portions of the nervous system when they occur concurrently with the afferent impulse arising from the action from some other stimulus energy than when the second afferent impulse is not occurring. Thus, two stimulus energies S_1 and S_2 when acting separately initiate afferent impulses s_1 and s_2 , but if acting at the same time these afferent impulses interact, changing each other to some extent so that by the time they reach the more central portions of the nervous system s_1 has changed to \tilde{s}_1 , and s_2 has changed to \tilde{s}_2 .

Now, suppose that S_1 and S_2 have each been conditioned separately to reaction, R . This process would produce the relationship,

$$S_1 \rightarrow s_1 \rightarrow r \rightarrow R$$

and

$$S_2 \rightarrow s_2 \rightarrow r \rightarrow R.$$

However, when S_1 and S_2 act simultaneously as a stimulus compound, the afferent impulse which tends to $r \rightarrow R$ in the one case is not s_1 but \tilde{s}_1 , and in the other case it is not s_2 but \tilde{s}_2 .

At this point the principle of afferent interaction in stimulus compounds is supplemented by the principle of the *primary stimulus generalization gradient* (Postulate 4, p. 178). In the present context this may be restated as follows: *The habit strength at the command of an afferent impulse is a decreasing growth function*

of the difference (d) between the evoking afferent impulse (\tilde{s}) and the afferent impulse (s) originally conditioned to the reaction. Thus the effective habit strength commanded by \tilde{s}_1 will be less than that commanded by s_1 as originally conditioned, i.e.,

$${}_1\tilde{H}_R < {}_1H_R.$$

From the two last mentioned principles there may be deduced a number of corollaries, some of which limit the generality of the corollaries just derived, and vice versa. Actually, all the principles here under discussion are operating in every hypothetical situation considered in the present chapter; in the interest of expository clarity their action is here taken up separately. They will be considered jointly in Chapter XIX (p. 349 ff.).

SOME COROLLARIES OF THE PRINCIPLE OF AFFERENT INTERACTION AND OF PRIMARY STIMULUS GENERALIZATION APPLIED TO STIMULUS COMPOUNDS

From the inequality last considered and the monotonic habit-reaction relationship it follows that,

$$R_{\tilde{s}_1} < R_{s_1}.$$

Generalizing, we arrive immediately at our fourth corollary:

IV. *If a stimulus aggregate has been conditioned to a reaction, and if, later, this stimulus aggregate is presented to the subject in conjunction with an alien stimulus aggregate not hitherto conditioned to the reaction in question, the strength of the reaction tendency evoked by the stimulus combination will be less than will that evoked by the stimulus aggregate as originally conditioned.*

This corollary is exemplified in one form of what Pavlov called *external inhibition*, i.e., the form where the "extra" stimulus which produces the external inhibition does not itself evoke a reaction conflicting with that normally evoked by the conditioned stimulus. In this connection Pavlov remarks (6, p. 45):

If one experimenter had worked with a dog and established some firm and stable conditioned reflex, conducting numerous experiments with them, when he handed the animal over to another experimenter to work with, all the reflexes disappeared for a considerable time. The same thing happened when the dog was changed over from one research room to another.

The interpretation is that the change in the stimulus situation produced by the presence of a new experimenter or by a different

room so modified the afferent impulses arising from the original conditioned stimulus that its reaction evocation power sank below the reaction threshold (3, p. 78).

What appears to be the same thing but in an even clearer form has been reported by Lashley (5, pp. 140-141). Rats were trained to jump to a black card bearing a white triangle and not to jump to a similar card bearing a white cross of the same area. After the animals had attained more than 95 per cent of correct choices, new cards were substituted which contained the same figures, with four small figures added. The score of "correct" choices with the new cards fell to 90 per cent. The interpretation is that the four additional figures so changed the afferent impulses arising from the triangle and the cross that the effective habit strength at the command of each was appreciably weakened, which naturally decreased the accuracy of the discrimination. It would appear that this secondary principle (external inhibition) is operating on a very large scale in all the higher organisms including ourselves, with whom it is often called "distraction of the attention."

A variant of the situation just considered is that in which two stimuli have been conditioned to the same reaction separately and then are presented to the organism simultaneously. The interaction of the two afferent impulses upon each other when occurring concurrently may be expected to reduce the effective habit strength of each so that the amplitude of the reaction evoked by them jointly will be appreciably less than would result from the simple summation of the two habit strengths. Thus suppose that S_1 and S_2 have each been conditioned separately to R to the extent of 70 habs, and that the afferent interaction effect of each on the other is of an extent sufficient to change the effective habit strength of $\bar{S}_1 H_R$ and $\bar{S}_2 H_R$ from 70 to 40 habs each. Now, the physiological summation of two habit strengths of 70 habs each yields 91 habs, whereas the summation of two habit strengths of 40 habs each yields 64 habs. Accordingly, the summation mechanism alone would reduce the effective habit strength of the two stimuli to 91 habs, and the interaction mechanism would reduce it still further—from 91 habs to 64 habs, which is less than the original value of the habit strength commanded by each individual component. It is evident that if the interaction effects are sufficiently great they may completely neutralize any summative effects otherwise resulting from combining homogeneous conditioned stimuli, and even yield a weaker reaction tendency than that evoked by either stim-

ulus aggregate acting alone. Our fifth corollary accordingly is as follows:

V. *If two stimuli separately and equally conditioned to the same reaction are later presented simultaneously, and their afferent neural interaction effects are sufficiently great, they will jointly evoke a weaker reaction tendency than would be mediated by either one of the stimuli acting alone.* Corollary V, it will be observed, constitutes a limitation on the generality of Corollaries I and II, the extent of the limitation depending on the magnitude of the afferent interaction involved in the change of stimulus conditions.

An experimental example of some historical interest illustrates this point. Shepard and Fogelsanger (7) had subjects learn paired nonsense syllables in the arrangement $A \rightarrow C$ and $B \rightarrow C$. Later, syllable A and syllable B were presented together. It was found that instead of decreasing the reaction latency of the evocation of syllable C (which would result from the physiological summation of two habit strengths), the joint presentation actually increased the weighted mean latency from 1135 milliseconds to 2638 milliseconds, the two latencies yielding a ratio of 1 to 2.323.

In the light of Corollary V these results present no paradox whatever. However, in some quarters they have produced a certain amount of confusion in the interpretation of rote-learning phenomena. For example, Köhler (4, p. 316) remarks of this particular experiment,

If it were not for organization one should expect that, both excitants working in the same direction, the syllable associated with them would be more easily reproduced than a syllable for which there was only one excitant. But the contrary was observed; it seemed as though some inhibition were in the way of reproduction when it was aroused by two excitants.

According to the present view, no inhibition whatever is involved in the determination of the Shepard-Fogelsanger results, and the unspecified organization or configurational factor is in reality a combination of afferent neural interaction and stimulus generalization.

A sixth corollary arises from a reversal of the situation presented by Corollary V. Suppose that two equally potent stimulus aggregates, S_1 and S_2 , have been conditioned jointly to the same reaction to the extent of 65.1 habs, and that later S_1 is presented to the subject separately. Since (Table 1) 65.1 habs corresponds to ten standard reinforcements, simple habit summation dynamics

would give S_1 the equivalent of five reinforcements, or 40.95 habs. Now, the interaction between afferent impulses arising from S_1 and S_2 will change what otherwise would have been s_1 to \tilde{s}_1 . Since the reaction is actually conditioned to \tilde{s}_1 (rather than to s_1), it follows that later, when S_1 is presented separately from S_2 and other dynamic stimuli, the afferent impulse thus sent into the central nervous system will be s_1 , rather than \tilde{s}_1 . But by the stimulus generalization gradient, s_1 will command a weaker effective habit strength than will \tilde{s}_1 , and so will evoke a weaker reaction tendency than is proper for a habit strength of 40.95 habs, depending upon the amount of afferent interaction which occurred in the original conditioning situation. If the interaction effects are of sufficient intensity, the effective habit strength at the command of one of the stimulus aggregates under the assumed conditions may not be greater than half that of the original stimulus compound S_1 and S_2 ; indeed, it may be even less. Our sixth corollary is accordingly formulated as follows:

VI. *If two stimulus aggregates, jointly and equally conditioned to a reaction, are later presented separately, the reaction strength evokable by each may be less than habit summation dynamics would indicate, and in extreme cases may be even less than half that of the compound originally conditioned, depending upon the amount of afferent neural interaction effects in the original conditioning situation.* It will be noted that Corollary VI also constitutes a limitation on the generality of Corollaries I and II, the extent of the limitation depending on the amount of afferent interaction involved.

Presumptive critical illustrations of Corollary VI are found in those cases of what Pavlov calls the "overshadowing" of one stimulus component in a conditioned stimulus compound by another component (6, pp. 142-144). For example, in one case a stimulus compound made up of a tone and three electric lamps was conditioned to the salivary reaction so that the compound would evoke eight drops of saliva in 30 seconds, though the lamps acting alone evoked no secretion whatever. On the above assumptions the lamps might very well have contributed materially to the joint reaction of eight drops, yet have been so weakened by the influence of afferent interaction under which conditioning took place as not to pass the reaction threshold when presented separately. The present set of hypotheses also demand that the tone when presented alone should have evoked less than eight drops in 30 sec-

onds; unfortunately Pavlov does not give the results of this control test.

SUMMARY

Some writers, in an attempt to simplify the account of the learning process, have left the erroneous impression that the conditioned stimulus, e.g., as represented by S in the symbol ${}_sH_R$, is a simple or singular energy operating on one receptor end organ or, at most, on a small number of such end organs of a single sense mode. In actual fact an immense number of receptor end organs are involved in every conditioning situation, however much it may have been simplified by experimental methodology. Each stimulus "object" represents a very complex aggregate of more or less alternative potential stimulations, often extending into numerous receptor modes.

Presumably every receptor which discharges an afferent impulse during the conditioning process acquires an increment of habit strength at each reinforcement. There is much reason to believe, however, that the magnitude of the increments acquired by the different receptor organs or receptor-organ aggregates varies greatly. Among the factors which are believed to favor the acquisition of large increments are: the dynamic or changing state of the stimulus energy, the intensity of the stimulus energy, the nature or "mode" of the receptor stimulated, the relative rarity of the occurrence of the stimulus energy, and the chance that the stimulus energy may previously have been conditioned to some strongly emotional reaction.

Despite the great rôle that stimulus compounds play in adaptive behavior, no unique primary principles have been found to be operating. One major principle which seems to be active is that of the summation of the habit-strength loadings borne by the several stimulus elements or aggregates of a stimulus compound: The habit strengths borne by the several stimulus components summate to form a single effective habit strength which is equal to that which would be produced by a number of consecutive standard reinforcements equal to the sum of the number of reinforcements which would be required to produce the separate habit strengths borne by the several stimulus components. The action of the principle of habit-strength summation is associated with a second primary principle, the monotonic habit-reaction function. This is that the strength of a reaction tendency, other things equal, is a monotonic

increasing function of the effective strength of the habit, and so of the reaction potential (p. 226 ff.) mediating it (Postulate 15, p. 344 ff.).

From these principles follows the first corollary, that the strength of the reaction tendency evoked by two homogeneous conditioned stimulus aggregates when acting in a stimulus compound is less than the arithmetical sum of the two reaction tendencies evokable by the components when acting separately. A second corollary closely related to the first is that the joint reaction tendency evoked by two stimulus components is greater than that evokable by either component acting separately. Both of these corollaries find experimental illustration under certain conditions. A third corollary of the same group is to the effect that if a number of stimulus aggregates are equally conditioned to a reaction, and varying numbers of them later simultaneously act on the receptors of the organism, the greater the number of aggregate so acting, other things equal, the greater will be the strength of the reaction tendency thus evoked.

A third primary principle which, along with those just considered, is believed to be active in determining the dynamics of stimulus compounds, is that known as afferent neural interaction. This principle is to the effect that when afferent receptor discharges occur at about the same time, they interact, changing each other to varying degrees depending upon circumstances not as yet well known (Postulate 2). Associated with the interaction principle is the familiar principle of the primary stimulus generalization gradient (Postulate 5).

From these latter primary principles there follow some additional corollaries. One of these is: In case a conditioned stimulus aggregate is presented in a compound with a second or "extra" stimulus aggregate, this stimulus compound, other things equal, will evoke a weaker reaction tendency than will the stimulus aggregate originally conditioned. An example of the action of this principle is found in one type of Pavlovian external inhibition.

NOTES

The Equation for the Combination of the Habit Loadings Borne by the Components of a Conditioned Stimulus Compound When the Components Are Two in Number

as derived by Dr. D. T. Perkins from an equivalent of the combination of the habit-strength loadings borne by the elements of compound

conditioned stimuli, in connection with the combination of two generalized habit tendencies evokable by a particular stimulus compound (1). Recast in a form appropriate for the present context, this equation is:

$$r_{s_1 s_2} H_R = s_1 H_R + s_2 H_R - \frac{s_1 H_R \times s_2 H_R}{M} \quad (32)$$

where s_1 is one afferent element or aggregate of a conditioned stimulus compound and s_2 is another such element or aggregate of the same conditioned stimulus compound, H is the power of the stimulus, represented by s_1 or s_2 , when combined with suitable motivation to evoke the reaction (R), and M is the physiological limit of conditioning strength under the circumstances in which the conditioning occurred.

It may easily be shown that the above equation is a special case of Day's general equation (30) for combining any number of habit strengths (XII, p. 200).

An Example of the Combination of the Habit Strengths of Two Conditioned Stimulus Elements or Aggregates by Means of Perkins' Equation

In connection with Corollary V (p. 218) there was occasion to determine the combined habit strengths of two conditioned stimulus elements or aggregates delivered simultaneously as a stimulus compound, each element by hypothesis carrying a habit loading of 40 habs. Because the exposition in the main text is designed for non-mathematical readers, such combination values are usually found by means of a table based on the simple growth function. The same outcome could, however, have been secured in all cases by the use of Perkins' equation, as was actually done in the case of Corollary V. In that example $s_1 H_R = 40$ habs and also $s_2 H_R = 40$ habs. Accordingly, substituting these values in Perkins' equation (32), we have,

$$\begin{aligned} s_1 + s_2 H_R &= 40 + 40 - \frac{40 \times 40}{100} \\ &= 80 - \frac{1600}{100} \\ &= 80 - 16 \\ \therefore s_1 + s_2 H_R &= 64 \end{aligned}$$

The Difficulty of Applying the Habit-Summation Equations in Quantitative Detail to Concrete Behavior Situations

A minor difficulty in the way of applying the habit summation equations to concrete behavior situations lies in the fact (p. 253 ff.) that habit must be combined with a drive (D) or motivation before the stimulus can evoke a reaction. If, however, both habit strength and reaction-evocation potential are on a centigrade scale, and the drive is chosen in such a way that its function when combined multiplicatively with that of $s H_R$ is unity, no serious difficulty will arise. Since it will not be possible to take up the matter of motivation until a later chapter, nothing has been said about this, lest the reader be unnecessarily confused.

A major difficulty lies in the fact that a large number of the stimulus elements always present in any conditioning situation can never be under direct and ready control of the investigator. As a result they can neither be administered sepa-

rately from those deliberately employed by the investigator (and usually of primary interest to him) nor wholly eliminated from experimental situations in which the experimenter seeks to determine the habit strength of certain stimulus aggregates such as the light or the vibrator employed in the galvanic conditioning situation (2) discussed above. If we let the aggregate habit loading of these normally uncontrollable factors be represented by x , then instead of saying that the galvanic reaction evoked by the light alone averaged 2.2 millimeters, we should say that the action evoked by the light *together with x unspecified and unmeasured elements in the conditioned stimulus situation* averaged 2.2 millimeters. In a similar manner, we should say that the conditioned galvanic skin reaction evoked by the joint action of the light, the vibrator, and *x unspecified and unmeasured stimulus elements* averaged 5.1 millimeters. It accordingly comes about that in the formula $\frac{R_1}{R_1 + R_2}$ the x -value appears twice in the denominator but only once in the numerator, *thus giving a smaller value to the ratio than it properly should have.*

It might be supposed that the habit loading of these x stimulus elements could be calculated by means of Day's equation (XII, p. 200). As a matter of fact this probably would be possible if we knew the value of the constant, M . On the other hand, the value of M could be calculated if we only knew the habit loading of the x stimulus elements. Perhaps the most promising possibility of escaping from this dilemma lies in the determination of the value of the constant M by some quite independent procedure. Meanwhile a considerable number of quasi-quantitative but empirically testable theorems may be derived from the equations even under present conditions. Three of these have been outlined above as corollaries based mainly on the hypothesis upon which both equations depend.

Finally, the conjecture may be appended that possibly these x stimulus elements, such as the cutaneous stimulations normally resulting from the contact of the organism with its support and the multitude of proprioceptive stimulus elements resulting from the posture of the organism, possess a relatively weak capacity for acquiring conditioned habit loadings owing to the fact that since they are more or less ubiquitous they must become conditioned in every conditioning situation the organism encounters. Unless these situations become very highly patterned on the stimulus side, it follows that stimulus elements which are conditioned to all kinds of reactions would ultimately become permanently extinguished and thus finally immune to any further conditioning. This of course would hold for usual or customary postures of the organism, but not for rare or unusual postures. There is great need for experimental research in this field, but the problem is a difficult one.

REFERENCES

1. HULL, C. L. The problem of stimulus equivalence in behavior theory. *Psychol. Rev.*, 1939, 46, 9-30.
2. HULL, C. L. Explorations in the patterning of stimuli conditioned to the G.S.R. *J. Exper. Psychol.*, 1940, 27, 95-110.
3. HULL, C. L. Conditioning: Outline of a systematic theory of learning. Chapter II in *The psychology of learning* (forty-first yearbook, National Society for the Study of Education). Bloomington, Ill.: Public School Publishing Co., 1942.

4. KÖHLER, W. *Gestalt psychology*. New York: Liveright, 1929.
5. LASHLEY, K. S. The mechanism of vision: XV. Preliminary studies of the rat's capacity for detail vision. *J. Gen. Psychol.*, 1938, 18, 123-193.
6. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
7. SHEPARD, J. F., and FOGELSANGER, H. M. Studies in association and inhibition. *Psychol. Rev.*, 1913, 20, 290-311.

CHAPTER XIV

Primary Motivation and Reaction Potential

It may be recalled that when the problem of primary reinforcement was under consideration (p. 68 ff.), the matter of organic need played a critical part in that the reduction of the need constituted the essential element in the process whereby the reaction was conditioned to new stimuli. We must now note that the state of an organism's needs also plays an important rôle in the causal determination of which of the many habits possessed by an organism shall function at a given moment. It is a matter of common observation that, as a rule, when an organism is in need of food only those acts appropriate to the securing of food will be evoked, whereas when it is in need of water, only those acts appropriate to the securing of water will be evoked, when a sexual hormone is dominant only those acts appropriate to reproductive activity will be evoked, and so on. Moreover, the extent or intensity of the need determines in large measure the vigor and persistence of the activity in question.

By common usage the initiation of learned, or habitual, patterns of movement or behavior is called *motivation*. The evocation of action in relation to secondary reinforcing stimuli or *incentives* will be called *secondary motivation*; a brief discussion of incentives was given above (p. 131) in connection with the general subject of amount of reinforcement. The evocation of action in relation to primary needs will be called *primary motivation*; this is the subject of the present chapter.

THE EMPIRICAL RÔLES OF HABIT STRENGTH AND DRIVE IN THE DETERMINATION OF ACTION

Casual observations such as those cited above often give us valuable clues concerning behavior problems, but for precise solutions, controlled quantitative experiments usually are necessary. In the present context we are fortunate in having an excellent empirical study which shows the functional dependence of the persistence of food-seeking behavior jointly on (1) the number of reinforcements of the habit in question, and (2) the number of

hours of food privation. Perin (12) and Williams (20) trained albino rats on a simple bar-pressing habit of the Skinner type (p. 87), giving separate groups different numbers of reinforcements varying from 5 to 90 under a standard 23 hours' hunger. Later the groups were subdivided and subjected to experimental

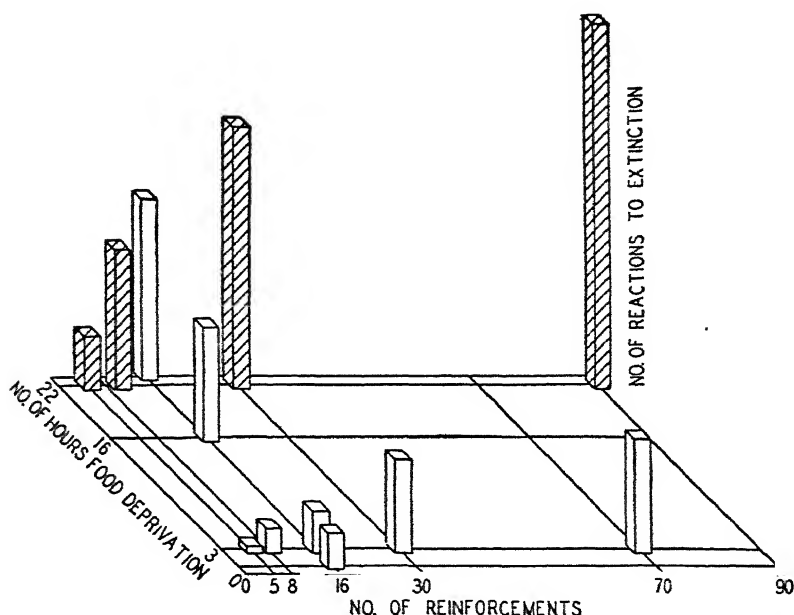


FIG. 48. Column diagram of the Perin-Williams data showing quantitatively how the resistance to experimental extinction in albino rats varies jointly with the number of reinforcements and the number of hours of food privation at the time the extinction occurred. The cross-hatched columns represent the groups of animals reported by Williams (20); the non-hatched columns represent the groups reported by Perin. (Figure reproduced from Perin, 12, p. 106.)

extinction¹ with the amount of food privation varying from 3 to 22 hours.

The gross outcome of this experiment is shown in Figure 48, where the height of each column represents the relative mean number of unreinforced reactions performed by each group before experimental extinction yielded a five-minute pause between successive bar pressures. The positions of the twelve columns on the base shows clearly the number of reinforcements and the number

¹ For an account of experimental extinction, see pp. 258 ff.

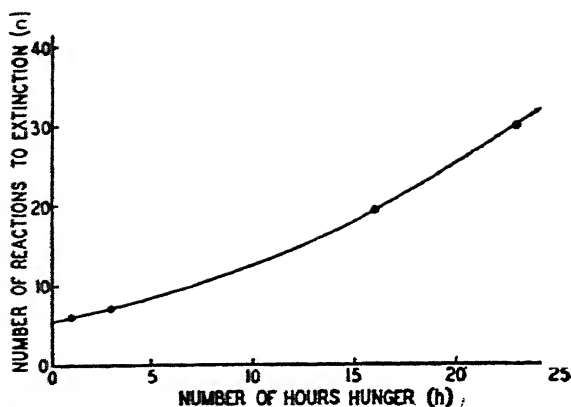


FIG. 49. Graphic representation of the data showing the systematic relationship between the resistance to experimental extinction (circles) and the number of hours' food deprivation where the number of reinforcements is constant at 16. The smooth curve drawn through the sequence of circles represents the slightly positively accelerated function fitted to them. This function is believed to hold only up to the number of hours of hunger employed in the original habit formation process: in the present case, 23. (Figure adapted from Perin, 12, p. 104.)

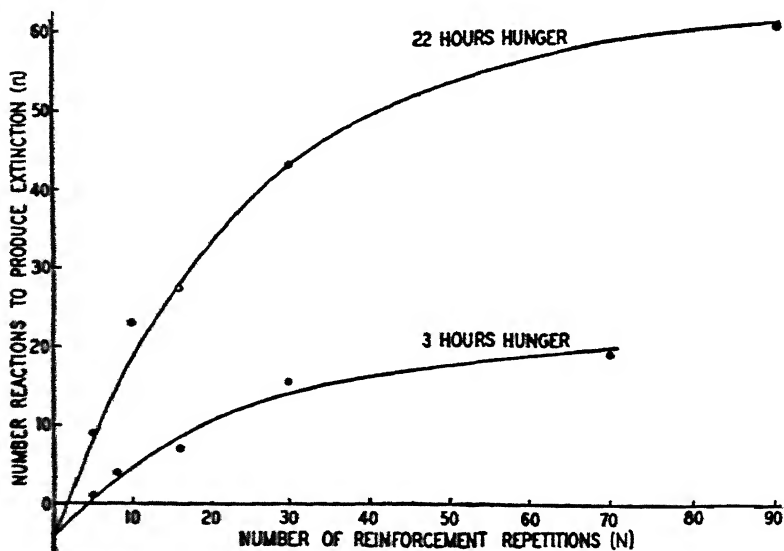


FIG. 50. Graphic representation of the two "learning" curves of Figure 48, shown in the same plane to facilitate comparison. The solid circles represent the empirical values corresponding to the heights of the relevant columns of Figure 48; the one hollow circle represents a slightly interpolated value. The smooth curves drawn among each set of circles represent the simple growth functions fitted to each set of empirical data. (Figure adapted from Perin, 12, p. 101.)

of hours' food privation which produced each. It is evident from an examination of this figure that *both* the number of reinforcements and the number of hours of food privation are potent factors in determining resistance to experimental extinction. Moreover, it is clear that for any given amount of food privation, e.g., 3 or 22 hours, the different numbers of reinforcements yield a close approximation to a typical positive growth function. On the other hand,

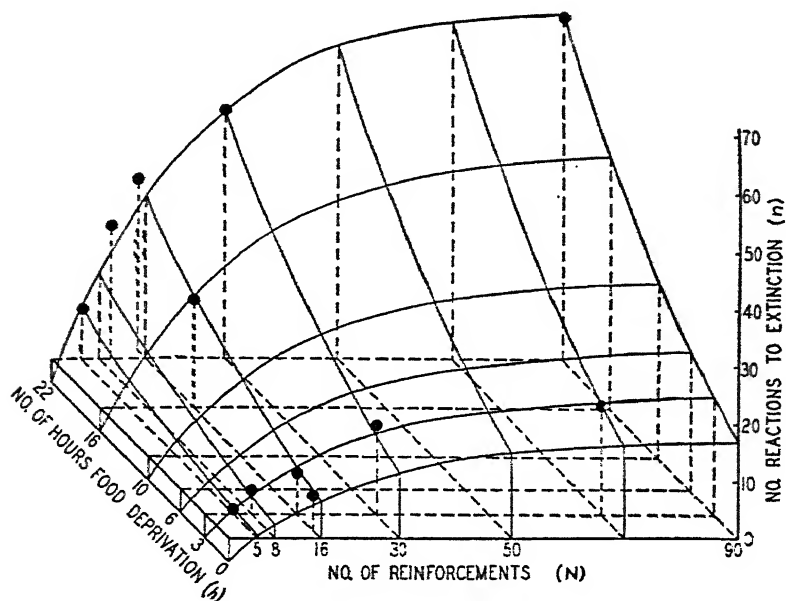


FIG. 51. Three-dimensional graph representing the fitted "surface" corresponding quantitatively to the action of the number of reinforcements and the number of hours of food privation following satiation, in the joint determination of the number of unreinforced acts of the type originally conditioned which are required to produce a given degree of experimental extinction. (Figure adapted from Perin, 12, p. 108.)

it is equally clear that for a given number of reinforcements, e.g., 16, the number of hours of food privation has an almost linear functional relationship to the resistance to experimental extinction.

For a more precise analysis of these functional relationships it is necessary to fit two-dimensional curves to the data. The results of this procedure are presented in Figures 49 and 50. Figure 49 shows that resistance to extinction at the 16-reinforcement level is a slightly positively accelerated function of the number of hours' food privation for the first 22 hours. Figure 50 shows that a posi-

tive growth function fits both "learning" curves fairly well. An examination of the equations which generated these curves reveals that the asymptotes differ radically, clearly being increasing func-

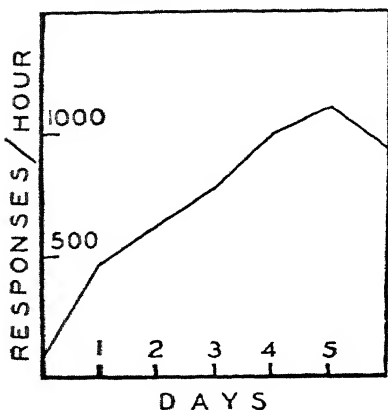


FIG. 52. Graph showing the relationship of the action potentiality as a function of the length of food privation following satiation. First note the fact that there is an appreciable amount of action potentiality at the beginning of this graph, where the amount of food privation is zero. Next, observe that the curve is relatively high at one day of food privation, which was the degree of drive under which the original training occurred. Finally, note that the rise in action potentiality is fairly continuous up to about five days, after which it falls rather sharply. This fall is evidently due to exhaustion, as the animals died soon after. The function plotted as the smooth curve of Figure 49 corresponds only to the first section of the present graph and clearly does not represent the functional relationship beyond a point where the number of hours of food privation is greater than 23. (Figure reproduced from Skinner, 13, p. 396.)

tions of the number of hours of food privation, but that the rates at which the curves approach their respective asymptotes are practically identical (F equals approximately $1/25$ in both cases). Finally it may be noted that both curves, when extrapolated backward to where the number of reinforcements would equal zero, yield a *negative* number of extinctive reactions amounting to approximately four. This presumably is a phenomenon of the reaction threshold which will be discussed in some detail later (p. 322); it is believed to mean that a habit strength sufficient to resist four extinction reactions is necessary before reaction will be evoked by the stimuli involved.

For a final examination of the outcome of the experiment as a whole, the curves shown in Figures 49 and 50 were synthesized in such a way as to yield a surface fitted to the tops of all the columns of Figure 48. This surface is shown in Figure 51. An examination of this figure reveals the important additional fact that when

the surface is extrapolated to where the number of hours' food privation is zero, the resistance to experimental extinction presumably will still show a positive growth function with n -values of considerable magnitude. As a matter of fact, the asymptote of the growth

function where $h = 0$ (satiation) is 28 per cent of that where $h = 22$ hours.

These last results are in fairly good agreement with comparable values from several other experimental studies. Measurements of one of Skinner's published graphs, reproduced as Figure 52, indicate that his animals displayed approximately 17 per cent as much food-seeking activity at satiation as at 25 hours' food privation. Finch (3) has shown that at satiation a conditioned salivary reaction in nine dogs yielded a mean of 24 per cent as much secretion as was yielded at 24 hours' food privation. Similarly, Zener (22) reports that the mean salivary secretion from four dogs average at satiation 24 per cent as much as at from 21 to 24 hours' food privation. The considerable amounts of responsiveness to the impact of conditioned stimuli when the organism is in a state of food satiation may accordingly be considered as well established.

The continued sexual activity of male rats for some months after castration points in the same direction. Stone (15) reports that male rats which have copulated either shortly before or shortly after castration, when an adequate supply of hormone would be present, continue to show sexual behavior sometimes as long as seven or eight months after removal of the testes. According to Moore *et al.* (10), Stone (15), and Beach (1), this operation removes within 20 days not only the source of testosterone but, through the resulting atrophy of accessory glands, also the source of other specifically supporting secretions. A few weeks after castration, therefore, when the normal supply of sex hormones in the animal's body has been exhausted, the sex drive is presumably in about the same state as is the food drive after complete food satiation. The continued sexual activity of these animals thus presents a striking analogy to the continued operation of the food-release bar by Perin's rats after food satiation. While not absolutely convincing, this evidence from the field of sexual behavior suggests that the performance of learned reactions to moderate degrees in the absence of the specific drive involved in their original acquisition may be sufficiently general to apply to all primary motivational situations.

Closely related to this same aspect of Perin's investigation is a study reported by Elliott (2). Albino rats were trained in a maze under a thirst drive with water as the reinforcing agent until the true path was nearly learned, when the drive was suddenly shifted to hunger and the reinforcing agent to food. The outcome of this

procedure is shown in Figure 53. There it may be seen that on the first trial under the changed condition of drive there was an appreciable disturbance of the behavior in the form of an increase in locomotor time; there was also an increase, of about the same proportion, in blind-alley entrances. On the later trials, however,

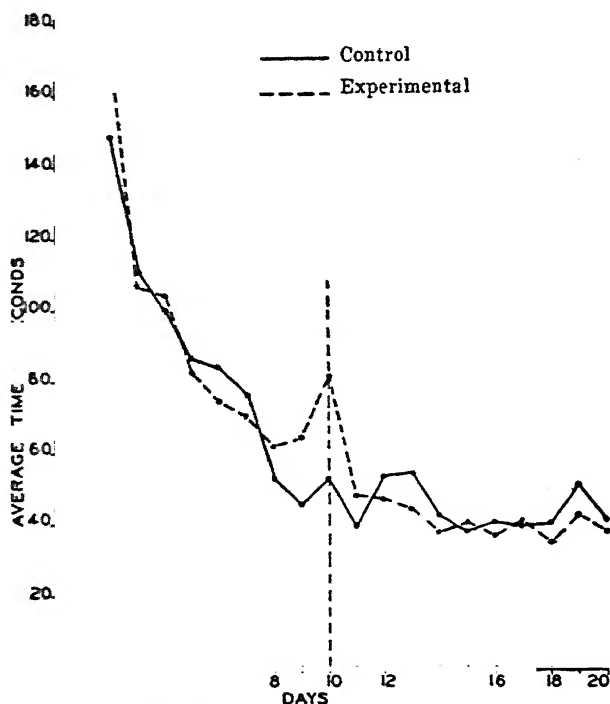


FIG. 53. Graphs showing the disruptive influence on a maze habit set up in albino rats on the basis of a water reinforcement, of having the drive (on the tenth day) suddenly shifted from thirst to hunger. (Reproduced from Elliott, 2, p. 187.)

the learning process appeared to proceed much as if no change had been made in the experimental conditions.

As a final item in this series there may be mentioned an empirical observation of Pavlov concerning the effect on an extinguished conditioned reaction of increasing the drive. On the analogy of Perin's experiment, it might be expected that this would again render the reaction evocable by the stimulus; and this in fact took place. In this connection Pavlov remarks (11, p. 127):

To illustrate this last condition we may take instances of differential inhibitions established on the basis of an alimentary reflex. If, for example, the dog has been kept entirely without food for a much longer period than usual before the experiment is conducted, the increase in excitability of the whole alimentary nervous mechanism renders the previously established differential inhibition wholly inadequate.

EMPIRICAL DIFFERENTIAL
REACTIONS TO IDENTICAL
EXTERNAL ENVIRONMENTAL
SITUATIONS ON THE BASIS
OF DISTINCT DRIVES

A second important type of motivational problem was broached in an experiment reported by Hull (6). Albino rats were trained in the rectangular maze shown in Figure 54. On some days a given animal would be run in the maze when satiated with water, but with 23 hours' food deprivation, whereas on other days the same rat would be run when satiated with food but with 23 hours of water deprivation. The two types of days alternated according to a predetermined irregularity. On the food-deprivation days the reinforcement chamber always contained food and the left entrance, say, to the chamber was only by traversing the right-hand water-deprivation days the reinforcement chamber always contained water, and the right-hand entrance would be blocked so that access

traversing the left-hand side of the rectangle. The outcome of this experiment is shown in Figure 55. There it may be seen that while learning was very slow, the animals of the experimental group gradually attained a considerable power of making the reaction which corresponded to the drive dominant at the time.

The capacity of rats to learn this type of discrimination was later demonstrated more strikingly by Leeper (8), in a substantially similar investigation. Leeper's experiment differed, however, in the

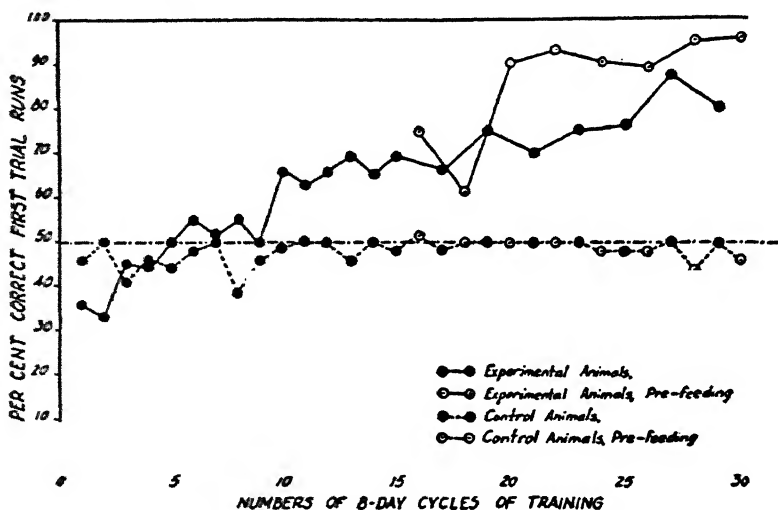


FIG. 55. Composite graphs showing the per cent of correct choices at the first trial of each experimental day in the discrimination by rats between hunger and thirst motivation (6, p. 263).

detail that two distinct reinforcement chambers were employed and no passageways were blocked at any time, so that if on a "food" day the rat went to the water side he always found water, and if on a "water" day he went to the food side, he always found food. Under these conditions the animals learned to perform the motivational discrimination with great facility; Leeper's animals needed only about one-twelfth the number of trials required by the original Hull technique, though again the process of acquisition was gradual.¹

¹This striking difference is attributed in part to the operation of spatial orientation and in part to the fact that when rats are deprived of either food or water they do not consume a normal amount of the other sub-

DOES THE PRINCIPLE OF PRIMARY STIMULUS-INTENSITY GENERALIZATION APPLY TO THE DRIVE STIMULUS (S_D)?

A factor with considerable possible significance for the understanding of motivation is the relationship between the degree of similarity of the need at the time of reinforcement and that at the time of extinction, on the one hand, and the associated resistance to experimental extinction on the other. No specific experiments have been found bearing exactly on this point, but several incidental and individually inconclusive bits of evidence may be mentioned as indicating the general probabilities of the situation.

The first of these was reported by Heathers and Arakelian (4). Albino rats were trained to secure food pellets by pressing a bar in a Skinner-Ellson apparatus. Next, half of the animals were partially extinguished under a weak hunger, and the remainder were extinguished to an equal extent under a strong hunger. Two days later the animals were subjected to a second extinction, half of each group under the same degree of hunger as in the first extinction, and the remaining half under a drive equal to the first-extinction hunger of the other group. Combining the state of food privation of the first and second extinctions, there were thus four hunger-extinction groups:

- 1, strong-strong; 2, strong-weak; 3, weak-strong; 4, weak-weak.

The authors report that a statistical pooling of the results from these four groups of animals revealed a tendency of the rats extinguished twice on the same drive to resist extinction less than did those animals which were extinguished the second time on a drive different from that employed on the first occasion. In two independent studies this difference amounted to approximately 4 and 6 per cent respectively; the latter results are reported to have a probability of 8 in 10 that the difference was not due to chance. This experimental outcome is evidently related to the primary generalization of stimulus intensity and suggests that *perseverative*

stance; this prevents genuine satiation of the supposedly satiated drive. For example, thirsty rats supposedly satiated with food will, after receiving even a few drops of water, very generally eat if food is available (8, p. 270); and rats, like humans, frequently drink while eating dry food if water is available. Thus after the first trial Leeper's animals were presumably operating under both drives, and one drive or the other was reinforced no matter which path was traversed.

extinction effects are to some extent specific to the primary drive or need intensity under which the extinction occurs.

By analogy, the stimulus-intensity generalization gradient apparently found in the case of extinction effects just considered strongly suggests the operation of the same principle in the case of reinforcement effects. Now, such a gradient has been demonstrated experimentally by Hovland (see p. 186 ff.); it naturally has its greatest value (Figure 43) at the point of reinforcement. Consequently it is to be expected that in a curve of motivation intensity such as that of Skinner (shown in Figure 52) a special elevation or inflection would appear at the drive intensity at which the original reinforcement occurred. Whether a mere coincidence arising from sampling errors or not, exactly such an inflection may be seen in Skinner's empirical graph at one day of food privation, which was in fact the drive employed by Skinner in the training of the animals in question. The present set of assumptions implies that if Skinner's curve as shown in Figure 52 were to be plotted in detail by hours rather than days, it would present a positive acceleration from zero to one day of food privation. Now, Perin's study did plot this region in some detail, and Figure 49 shows that a positively accelerated function was found. These facts still further increase the probability that the principle of stimulus-intensity generalization applies to the drive stimulus (S_D).

THE INFLUENCE OF CERTAIN DRUGS ON EXPERIMENTAL EXTINCTION AND ITS PERSEVERATIONAL EFFECTS

Certain drugs are known to influence markedly the phenomena of experimental extinction. Switzer (18) investigated the effect of caffeine citrate on the conditioned galvanic skin reaction in human subjects, using a control dose of milk sugar. He found that caffeine increased resistance to experimental extinction; incidentally he also found that caffeine increased the amplitude of the unconditioned galvanic skin reaction and decreased the reaction latency.

Pavlov (11, p. 127) reported a somewhat related experiment performed by Nikiforovsky. An alimentary salivary conditioned reflex had been set up to a tactile stimulus on a dog's forepaw. This reaction tendency generalized to other parts of the animal's skin, including a point on the back which subsequently was completely extinguished. At the latter stage of training the stimulus on the paw yielded five drops of saliva during the first minute of

stimulation, whereas stimulation of the extinguished spot on the back yielded a zero reaction. Thereupon, the animal was given a subcutaneous injection of 10 c.c. of 1 per cent solution of caffeine. A few minutes later the stimulus when applied to the forepaw evoked four drops during the first minute, and when applied to the previously extinguished spot on the back, yielded three drops (11, p. 128), thus indicating a major dissipation of the extinction effects.

Miller and Miles (9) have contributed to this field. They demonstrated in albino rats traversing a 25-foot straight, enclosed runway that an injection of caffeine sodio-benzoate reduced the locomotor retardation due to experimental extinction by about two-thirds. In the same study it was shown that the retardation in locomotor time due to satiation was reduced by the caffeine solution approximately one-half (9).

Benzedrine is another substance which when thrown into the blood stream has the power of greatly retarding the onset of experimental extinction. This was demonstrated by Skinner and Heron (14) to hold for the Skinner bar-pressing habit.

SEX HORMONES AND REPRODUCTIVE ACTIVITY

As a final set of empirical observations concerning motivation we must consider briefly the relation of sex hormones to reproductive behavior. Within recent years an immense amount of excellent experimental work has been performed in this field, though only brief notice of it can be taken in this place. An account of two typical bits of this work was given above (Figures 11 and 12). In a recent comprehensive summary by Beach (1) the following propositions appear to have fairly secure empirical foundation:

1. Animals of practically all species which through castration have become sexually unresponsive to ordinary incentive stimulation, become responsive promptly on the injection of the appropriate hormone—usually testosterone propionate for males and estrogen for females.

2. Presumptively normal male rats differ greatly in their sexual responsiveness, all the way from those which will attempt copulation with inanimate objects to those which will not react even to an extremely receptive and alluring female. The injection of testosterone usually raises the reactivity of all but a few of the most sluggish animals. Alternatively, the presentation of an especially attractive incentive tends to have the same objective effect, though to a lesser degree (17).

3. Destruction of the cerebral cortex decreases sexual reactivity roughly in proportion to the extent of such destruction, very much as occurs in

the case of food habits. If destruction has not been too great, injection of the hormone will largely restore sexual responsiveness to appropriate incentives. The presentation of an exceptionally attractive incentive will, however, have much the same effect upon the objective behavior of such organisms.

4. Virgin male organisms which are unresponsive to an ordinary receptive female, after a few copulations under the influence of an injection of the hormone will remain responsive long after the hormone has presumably disappeared from the animal's body. This is believed to be caused by the learning resulting from the incidental reinforcement which occurred when the animal was under the influence of the hormone (1).

5. Many intact individuals of both sexes in most species occasionally manifest a portion of the behavior pattern characteristic of the opposite sex. Injection of the sex hormone of the opposite sex in castrated individuals of either sex tends strongly to the evocation of the sexual behavior pattern characteristic of normal organisms of the opposite sex on appropriate stimulation; this, however, is not usually as complete as the gross anatomical equipment of the organisms would seem to permit. Curiously enough, large doses of testosterone given to male rats make possible the elicitation of all elements of the typical female sexual behavior (1).

PRIMARY MOTIVATIONAL CONCEPTS

With the major critical phenomena of primary motivation¹ now before us, we may proceed to the attempt to formulate a theory which will conform to these facts.

At the outset it will be necessary to introduce two notions not previously discussed. These new concepts are analogous to that of habit strength (sH_R) which, it will be recalled (p. 114), is a logical construct conceived in the quantitative framework of a centigrade system.

The first of the two concepts is *strength of primary drive*; this is represented by the symbol D . The strength-of-drive scale is conceived to extend from a zero amount of primary motivation (complete satiation) to the maximum possible to a standard organism of a given species. In accordance with the centigrade principle this range of primary drive is divided into 100 equal parts or units. For convenience and ease of recall, this unit will be called the *mote*, a contraction of the word *motivation* with an added *e* to preserve normal pronunciation.

Because of the practical exigencies of exposition the second of

¹The empirical phenomena of secondary motivation, including such matters as incentive (p. 121 ff.), fractional anticipatory goal- and subgoal-reactions, cannot be treated in the present volume because space is not available.

the new concepts has already been utilized occasionally in the last few pages, where it has been referred to as the "reaction tendency," a term in fairly general use though lacking in precision of meaning. For this informal expression we now substitute the more precise equivalent, *reaction-evocation potentiality*; or, more briefly, *reaction potential*. This will be represented by the symbol ${}_sE_R$. Like habit (${}_sH_R$) and drive (D), reaction-evocation potential is also designed to be measured on a 100-point scale extending from a zero reaction tendency up to the physiological limit possible to a standard organism. The unit of reaction potentiality will be called the *wat*, a contraction of the name *Watson*.

It should be evident from the preceding paragraphs that D and ${}_sE_R$ are symbolic constructs in exactly the same sense as ${}_sH_R$ (see p. 111 ff.), and that they share both the advantages and disadvantages of this status. The drive concept, for example, is proposed as a common denominator of all primary motivations, whether due to food privation, water privation, thermal deviations from the optimum, tissue injury, the action of sex hormones, or other causes. This means, of course, that drive will be a different function of the objective conditions associated with each primary motivation. For example, in the case of hunger the strength of the primary drive will probably be mainly a function of the number of hours of food privation, say; in the case of sex it will probably be mainly a function of the concentration of a particular sex hormone in the animal's blood; and so on. Stated formally,

$$D = f(h)$$

$$D = f(c)$$

$$D = \text{etc.,}$$

where h represents the number of hours of food privation of the organism since satiation, and c represents the concentration of a particular hormone in the blood of the organism.

Turning now to the concept of reaction-evocation potentiality, we find, thanks to Perin's investigation sketched above (p. 227 ff.), that we are able at once to define ${}_sE_R$ as the product of a function of habit strength (${}_sH_R$) multiplied by a function of the relevant drive (D). This multiplicative relationship is one of the greatest importance, because it is upon ${}_sE_R$ that the amount of action in its various forms presumably depends. It is clear, for example, that it is quite impossible to predict the vigor or persistence of a given type of action from a knowledge of either habit strength or

drive strength alone; this can be predicted only from a knowledge of the product of the particular functions of sH_R and D respectively; in fact, this product constitutes the value which we are representing by the symbol sE_R .

SUMMARY AND PRELIMINARY PHYSIOLOGICAL INTERPRETATION OF EMPIRICAL FINDINGS

Having the more important concepts of the systematic approach of primary motivation before us, we proceed to the formulation of some empirical findings as related to motivation.

Most, if not all, primary needs appear to generate and throw into the blood stream more or less characteristic chemical substances, or else to withdraw a characteristic substance. These substances (or their absence) have a selective physiological effect on more or less restricted and characteristic portions of the body (e.g., the so-called "hunger" contractions of the digestive tract) which serves to activate resident receptors. This receptor activation constitutes the drive stimulus, S_D (p. 72 ff.). In the case of tissue injury this sequence seems to be reversed; here the energy producing the injury is the drive stimulus, and its action causes the release into the blood of adrenal secretion which appears to be the physiological motivating substance.

It seems likely, on the basis of various analogies, that, other things equal, the intensity of the drive stimulus would be some form of negatively accelerated increasing function of the concentration of the drive substance in the blood. However, for the sake of expository simplicity we shall assume in the present preliminary analysis that it is an increasing linear function.

The afferent discharges arising from the drive stimulus (S_D) become conditioned to reactions just the same as any other elements in stimulus compounds, except that they may be somewhat more potent in acquiring habit loadings than most stimulus elements or aggregates. Thus the drive stimulus may play a rôle in a conditioned stimulus compound substantially the same as that of any other stimulus element or aggregate (p. 74 ff.). As a stimulus, S_D naturally manifests both qualitative and intensity primary stimulus generalization in common with other stimulus elements or aggregates in conditioned stimulus compounds (p. 185 ff.).

It appears probable that when blood which contains certain chemical substances thrown into it as the result of states of need,

or which lacks certain substances as the result of other states of need, bathes the neural structures which constitute the anatomical bases of habit ($_sH_R$), the conductivity of these structures is augmented through lowered resistance either in the central neural tissue or at the effector end of the connection, or both. The latter type of action is equivalent, of course, to a lowering of the reaction threshold and would presumably facilitate reaction to neural impulses reaching the effector from any source whatever. As Beach (1) suggests, it is likely that the selective action of drives on particular effector organs in non-learned forms of behavior acts mainly in this manner. It must be noted at once, however, that sensitizing a habit structure does not mean that this alone is sufficient to evoke the reaction, any more than that caffeine or benzedrine alone will evoke reaction. Sensitization merely gives the relevant neural tissue, upon the occurrence of an adequate set of receptor discharges, an augmented facility in routing these impulses to the reactions previously conditioned to them or connected by native (inherited) growth processes. This implies to a certain extent the undifferentiated nature of drive in general, contained in Freud's concept of the "libido." However, it definitely does not presuppose the special dominance of any one drive, such as sex, over the other drives.

While all drives seem to be alike in their powers of sensitizing *acquired* receptor-effector connections, their capacity to call forth within the body of the organism characteristic and presumably distinctive drive stimuli gives each a considerable measure of distinctiveness and specificity in the determination of action which, in case of necessity, may be sharpened by the process of patterning (see p. 349 ff.) to almost any extent that the reaction situation requires for adequate and consistent reinforcement. In this respect, the action of drive substances differs sharply from that of a pseudo-drive substance such as caffeine, which appears to produce nothing corresponding to a drive stimulus.

Little is known concerning the exact quantitative functional relationship of drive intensity to the conditions or circumstances which produce it, such as the number of hours of hunger or the concentration of endocrine secretions in the blood. Judging from the work of Warden and his associates (19), the relationship of the hunger drive up to two or three days of food privation would be a negatively accelerated increasing function of time, though a study by Skinner (Figure 52) suggests that it may be nearly linear

up to about five days. For the sake of simplicity in the present explorational analysis we shall assume the latter as a first approximation.

Physiological conditions of need, through their sensitizing action on the neural mediating structures lying between the receptors and the effectors (sH_R), appear to combine with the latter to evoke reactions according to a multiplicative principle, i.e., reaction-evocation potentiality is the product of a function of habit strength multiplied by a function of the strength of drive:

$$sE_R = f(sH_R) \times f(D).$$

In the next section it shall be our task to consider in some detail what these functions may be; if successful we shall then possess the main portion of a molar theory of primary motivation.

THE QUANTITATIVE DERIVATION OF sE_R FROM sH_R AND D

Since we have taken Perin's experiment as our main guide in the analysis of the primary motivational problem in general, it will be convenient to take the need for food as the basis for the detailed illustration of the working of the molar theory of motivation; this we now proceed to develop.

Turning first to the habit component of sE_R , we calculate the values of sH_R as a positive growth function; we use in this calculation the fractional incremental value (F) found by Perin to hold for the learning processes represented in Figure 50, which was approximately $1/25$ for each successive reinforcement. On this assumption the values at various numbers of reinforcements, e.g., 0, 1, 3, 9, 18, 36, and 72, have been computed. These are shown in column 2 of Table 5.

The habit-strength values of column 2, Table 5, consist of the physiological summation of the habit-strength loadings of the stimulus components, represented by the original drive stimulus S_D' and the non-drive components, which we shall represent by S_I . Assuming as a matter of convenience that S_D' and S_I have equal loadings, the value of each (see fifth terminal note) is easily calculated for the several numbers of reinforcements. These values are shown in column 3 of Table 5.

Turning next to the matter of drive, it will be assumed that the original learning took place under a 24-hour food privation. Assuming further that drive is a linear function of the number of

TABLE 5

TABLE SHOWING THE PRELIMINARY STEPS IN THE DERIVATION OF A SERIES OF THEORETICAL REACTION-POTENTIAL VALUES FROM A VARIED SET OF ANTECEDENT REINFORCEMENTS UNDER A DRIVE OF 20 UNITS' STRENGTH, THE RESULTING HABITS BEING EVALUATED FOR REACTION POTENTIALITY AT DRIVE-STRENGTHS OF 0.00, 2.50, 6.67, 13.33, AND 20.0 UNITS.

N	Habit Strength as Formed		Effective Habit Strength of Drive-Stimulus Component ($s_D \bar{H}_R$) Derived from Intensity Generalization Gradient					Physiological Summation of Habit Strengths of Both Components ($s_1 + s_D \bar{H}_R$)					Reaction-Potential (sE_R) as Mediated by Various Strengths of Drive				
	Two Components $s_1 + s_D \bar{H}_R$	One Component ($s \bar{H}_R$)	$d' = 20.00$	$d' = 17.5$	$d' = 13.33$	$d' = 6.67$	$d' = 0.00$	$D = 0.00$	$D = 2.50$	$D = 6.67$	$D = 13.33$	$D = 20.00$	$D = 0.00$	$D = 2.50$	$D = 6.67$	$D = 13.33$	$D = 20.00$
			4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1	3.05	1.54	1.03	1.08	1.18	1.35	1.54	2.55	2.60	2.70	2.87	3.06	0.23	.30	.41	.61	.83
3	8.77	4.49	3.00	3.15	3.43	3.92	4.49	7.36	7.50	7.77	8.23	8.78	.67	.85	1.18	1.75	2.39
9	23.35	12.46	8.31	8.75	9.54	10.89	12.46	19.73	20.12	20.81	21.99	23.37	1.70	2.29	3.15	4.66	6.37
18	39.43	22.18	14.81	15.58	16.95	19.38	22.18	33.71	34.30	35.37	37.26	39.44	3.06	3.90	5.36	7.90	10.76
36	58.13	35.29	23.56	24.78	26.96	30.84	35.29	50.54	51.33	52.74	55.25	58.13	4.59	5.83	7.99	11.72	15.85
72	71.21	46.34	30.94	32.54	35.40	40.50	46.34	62.04	63.80	65.34	68.07	71.21	5.72	7.25	9.90	14.44	19.42

hours' hunger and that (Figure 52) the maximum of 100 motes would be reached at five days or 120 hours, Perin's periods of food privation may be converted into units of drive strength by multiplying the number of hours' food privation by the fraction $100/120$. In this way we secure the following drive or D -values:

Number of hours' food privation (h):	0	3	8	16	24
Strength of drive in motes (D):	0	2.5	6.667	13.333	20
Deviation (d) of possible D 's from the drive (D') of original learning:	20	17.5	13.333	6.667	

Now, S_D is assumed to be approximately a linear function of D . It follows from this and the principle of primary stimulus generalization that action evoked under any other intensity of drive (and drive stimulus) than that involved in the original habit formation must be subject to primary intensity-stimulus generalization. Assuming the relatively flat gradient yielded by an F -value of $1/50$, it is easy to calculate the value of ${}_S D \bar{H}_R$ (p. 199 ff.) at each degree of the five D -values taken above. These ${}_S D \bar{H}_R$ values are shown in columns 4, 5, 6, 7, and 8 respectively of Table 5. A glance at the bottom entries of each of the columns shows that the values of ${}_S D \bar{H}_R$ fall progressively from 46.34 at $D = 20$ (i.e., $d = 0.00$) to 30.94 at $D = 0$ (i.e., $d = 20$).

We must now combine these habit values by the process of physiological summation characteristic of conditioned stimulus compounds (p. 223 ff.) (neglecting the effects of afferent interaction) with the habit loading of the non-drive stimulus component of the compound which is represented by the values appearing in column 3. The physiological summation of the values in column 3 with the values of columns 4 to 8 gives us the habit-strength values shown in columns 9, 10, 11, 12, and 13 of Table 5. It will be noticed that this final recombination of the ${}_S H_R$ values where $D = 20$ yields exactly the same values as those of column 2. This is because when reaction evocation occurs at the original drive (D'), i.e., where $D = D'$, no distortion of the S_D component of the habit results, the synthesis being exactly the reverse of the analysis which took place between columns 2 and 3.

With the theoretical values of $f({}_S H_R)$ available in columns 9 to 13 inclusive of Table 5, we may now turn our attention to the problem of $f(D)$. It is assumed that D itself acts upon ${}_S H_R$ as a direct proportion. However, there is the complication that other

or alien drives active at the time (represented in the aggregate by the symbol \dot{D}) have the capacity to sensitize habits not set up in conjunction with them. Let it be supposed that this generalized effect of alien drives adds 10 points to the actual drive throughout the present situation. Thus the effective drive (\bar{D}) operative on a given habit would necessarily involve the summation of \dot{D} and D ; in the case of the 24-hour food privation a simple summation would in the present situation amount to $10 + 20$, or 30, and at 120 hours it would be $10 + 100$, or 110. In order to maintain our centigrade system the simple summation must be divided by the maximum possible under these assumptions, or 110. Accordingly we arrive at the formula,

$$\bar{D} = 100 \frac{\dot{D} + D}{\dot{D} + 100}$$

where \bar{D} represents the *effective drive* actually operative in producing the reaction potential.

Now, assuming that reaction evocation potentiality is essentially a multiplicative function of habit strength and drive, i.e., that,

$${}_sE_R = f({}_sH_R) \times f(D),$$

since $f({}_sH_R)$ is ${}_s\bar{H}_R$, and $f(D)$ is \bar{D} , we have by substitution,

$${}_sE_R = {}_s\bar{H}_R \times \bar{D}.$$

However, since both ${}_s\bar{H}_R$ and \bar{D} are on a centigrade scale, their simple product would yield values on a ten-thousand point scale; therefore, to keep ${}_sE_R$ also to a centigrade scale we write the equation,

$${}_sE_R = \frac{{}_s\bar{H}_R \times \bar{D}}{100}$$

Substituting the equivalent of \bar{D} and simplifying, we have as our final equation,

$${}_sE_R = {}_s\bar{H}_R \frac{\dot{D} + D}{\dot{D} + 100}$$

The second portion of this formula, with the various D values substituted, is,

	$\frac{10 + 2.5}{110}$	$\frac{10 + 6.667}{110}$	$\frac{10 + 13.333}{110}$	$\frac{10 + 20}{110}$
.0909	.1136	.1515	.2121	.2727.

The values of sE_E are accordingly obtained simply by multiplying the several entries of column 9 by .0909, those of column 10 by .1136, and so on. These products are presented in detail in columns 14, 15, 16, 17 and 18 of Table 5, which are the values we have been seeking; they are shown diagrammatically by the curved surface of Figure 56. A comparison of the theoretical values of Figure 56 with the surface fitted to the empirical values represented

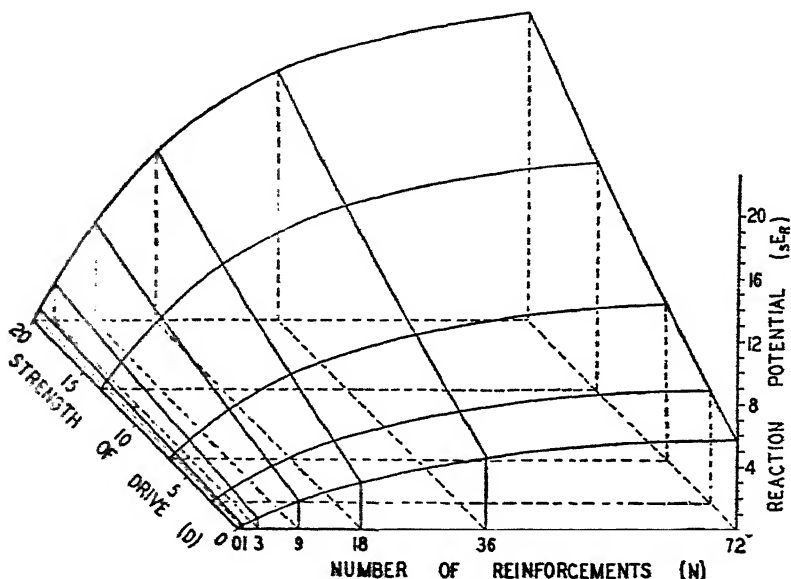


FIG. 56. Graphic representation of the theoretical joint determination of reaction potential by various numbers of reinforcements under a drive (D') of 20 units' strength when functioning under drives (D) of various strengths less than that of the original habit formation. Note the detailed agreement with the comparable empirical results shown in Figure 51.

by the circles in Figure 51 indicates that the theoretical derivations approximate the facts very closely indeed.

Computations analogous to the preceding have shown that the present set of postulates and constants also hold when $D > D'$ at least up to three days of food privation. The theoretical curve for all values of D between 0 and 72 hours yields a positively accelerated reaction potential up to 24 hours (D' in the present analysis), where there is a slight inflection; as D increases above D' there is at first a brief period of positive acceleration, which is

followed by a protracted period that is nearly linear, the whole showing a fair approximation to Figure 52.

Generalizing from Table 5 and Figure 56, the following corollaries may be formulated as a kind of condensed summary of the implications of the present set of assumptions as shown by the preceding computations:

I. *When habit strength is zero, reaction-evocation potential is zero.*

II. *When primary drive strength (D) is zero, reaction-evocation potential (${}_sE_R$) has an appreciable but relatively low positive value which is a positive growth function of the number of reinforcements. Corollaries I and II both agree in detail with Perin's empirical findings.*

As the drive (D) increases from zero to D' :

III. *The reaction-evocation potential increases with a slight positive acceleration.*

IV. *The reaction-evocation potential maintains its positive growth relationship to the number of reinforcements. Both of these corollaries agree in detail with Perin's empirical findings.*

As the drive (D) increases above D' :

V. *There is a definite inflection in the ${}_sE_R$ function at D , the slope for values of D just greater than D' being less than for those just below.*

VI. *The reaction-evocation potential above D' increases at first with a slight positive acceleration, which soon gives place to a practically linear relationship. Both of these corollaries agree in detail with Skinner's empirical findings (Figure 54).*

MISCELLANEOUS COROLLARIES FLOWING FROM THE PRESENT PRIMARY MOTIVATION HYPOTHESIS

The first problem in this series is that presented by Elliott's experiment described above, the outcome of which is clearly shown in Figure 53. Here we have the case of a reaction tendency set up on the basis of one drive, showing a partial but by no means complete disruption when this drive (thirst) is abruptly replaced by another drive, that of hunger. At this point we recall the assumption stated earlier (p. 241) that all drives alike are able to sensitize all habits. Applying this to the behavior of Elliott's animals during the first critical test trial on the maze after the change in drive, it is to be expected that while hunger was then the dominant

drive, certain residual amounts of various other drives (including thirst) were also active. These in the aggregate (\dot{D}), the hunger drive included, are presumed to operate in a multiplicative manner upon the habit strength effective at the moment in determining reaction potentiality. It is assumed that this would be enough to evoke, on the average, about 20 per cent as much activity as is evoked by the thirst drive.

This means that the residual drive (\dot{D}) must amount to considerably more than 20 per cent of the regular thirst drive, say. For example in the detailed analysis of the preceding section, where 24 hours' hunger stood at 20 units of drive, this residual drive was placed at 10 units, which is 50 per cent as much as 20. Nevertheless, the reaction potential at 24 hours' hunger came out at 19.42 units, whereas that at satiation or zero drive stood at 4.58 units, the latter being only about 23 per cent of the former. The explanation of the paradoxical difference of 50 versus 23 per cent is significant; it arises largely from the fact that when the hitherto dominant drive ceases to be active, not only are there lost the 20 units of drive strength previously contributed by this need, but *there is also lost to the conditioned stimulus compound the sizable component made up by S_D , the withdrawal of which materially reduces the available habit strength associated with the situation in question, and so reduces the resulting reaction potential.*

On the basis of the above analysis we may formulate the following additions to the corollaries listed in the preceding section:

VII. *Under the conditions of the satiation of the dominant drive involved in the original habit-acquisition, there are sufficient residuals of other drives which in the aggregate yield on the average an excitatory potential amounting to around 20 per cent of that mobilized by a 24-hour hunger on a habit originally set up on the basis of this drive.*

VIII. *In case an organism is presented with all the stimuli characteristic of a habit, if the original drive is replaced by a strong second drive whose S_D activates no conflicting habit tendency, the reaction potential to the execution of the habitual act will be stronger than would be the case if the irrelevant second drive were not active. This means that if a control group with both hunger and thirst thoroughly satiated were to be added to the Elliott experiment described above, the mean retardation in the running time and the mean number of errors would increase appreciably above what resulted from a mere replacement of one drive by another (6).*

A second problem concerns the relation of the experimental extinction of a reaction tendency to the drive intensity operative at the time of extinction. Now, the passage from Pavlov quoted above (p. 233) strongly suggests that *experimental extinction effects are in some sense directly opposed to reaction potential rather than merely to habit strength*. Since with a constant habit strength an increase in the drive augments the reaction potential, and since extinction effects are an increasing function of the number of unreinforced evocations, it follows that:

IX. *The number of reinforcements being constant, the stronger the relevant drive, the greater will be the number of unreinforced evocations which will be required to reduce the reaction potential to a given level.*

X. *The number of reinforcements being constant, the stronger an allied but irrelevant drive active at the time of extinction, the greater will be the number of unreinforced evocations required to reduce the reaction potential to a given level, though this number will be materially less than would be required under the same intensity of the relevant drive.*

Thus if a habit set up on the basis of a thirst drive were extinguished under a sizable hunger drive but with water satiation, the theory demands that the reaction potential would extinguish with fewer unreinforced evocations than would be the case under the same intensity of the thirst drive in conjunction with a zero hunger drive; moreover, such a habit would require more unreinforced reaction evocations to produce a given degree of extinction under a strong hunger drive than under a weak one. By the same type of reasoning it is to be expected that if a reaction tendency were set up in male rats under hunger or thirst, and if subsequently a random sample of the organisms were castrated, experimental extinction under a normal hunger drive would occur more quickly than it would in the non-castrated organisms.

At this point we turn to a more detailed consideration of Pavlov's observation just referred to, that when he had performed an experimental extinction under a given drive and then increased the drive, the conditioned stimulus would again evoke the reaction. This may be deduced rather simply: If a certain number of unreinforced evocations of a reaction have produced sufficient extinction effects to neutralize a given amount of excitatory potential, an increase in the drive will increase the excitatory potential which the existent extinction effects will no longer suffice to neutralize

completely. The balance of the reaction potential will accordingly be available to evoke reaction and, upon adequate stimulation, will do so. We thus come to our eleventh corollary:

XI. *If a reaction tendency is extinguished by massed reaction evocations under a given strength of drive, and if at once thereafter the drive is appreciably increased, the original stimulation will again evoke the reaction.*

Our final question concerns an exceedingly important problem in adaptive dynamics. It has already been pointed out that as a rule action sequences required to satisfy a food need are different from those required to satisfy a water need, and both would ordinarily be quite different from the acts which would be required to satisfy a sex drive. This problem is posed very sharply when, as in the Hull-Leeper experiments, an organism is presented with an identical objective situation and required to make a differential reaction purely on the basis of the need dominant at the moment. These experiments confirm everyday observations that animals can adapt successfully to such situations. The question before us is how this behavior is to be explained.

At first sight it might be supposed that in this situation the animals would merely associate S_k with turning to the right, say, and S_l with turning to the left, and that adaptation would thereby be complete. A little further reflection will show, however, that this simple explanation is hardly adequate, because if there were really an independent and functionally potent receptor-effector connection between the hunger-drive stimulus and turning to the right the animal would, when hungry, be impelled to turn to the right continuously when in its cage or wherever it happened to be, as well as at the choice point in the maze. The animals, of course, display no such behavior, any more than we ourselves do.

The present set of postulates mediates the explanation chiefly on the basis of a secondary process known as *patterning*. Unfortunately it will not be possible to give an exposition of this exceedingly important subject until a later chapter (see p. 349 ff.). However, pending the detailed presentation in that place we shall here merely indicate dogmatically the nature of patterning and briefly sketch the application of this secondary principle to the problem in adaptive dynamics now before us.

By the term "patterning" we mean the process whereby organisms acquire the capacity of reacting (or not reacting) to particular combinations of stimuli as distinguished from the several com-

ponent stimulus elements or aggregates making up the compound. At bottom this process turns out to be a case of learning to discriminate afferent interaction effects (p. 42 ff.). Specifically, the principle of afferent interaction implies that in the Hull-Leeper studies afferent impulses (\tilde{S}) arising from the environmental stimuli (S_i) are somewhat different when stimulation occurs in combination with the hunger-drive stimulus (S_h) from those which result from the same stimulation in combination with the thirst-drive stimulus (S_t). Similarly, the afferent impulses arising from S_h and S_t are somewhat different when initiated in conjunction with S_i from those initiated by S_h and S_t in the cage or other situations. If the afferent impulses arising from the environmental stimuli uncomplicated by any particular drive be represented by s , then these impulses when modified by the interaction with the hunger-drive stimulus may be represented by \tilde{s}_h , and when modified by interaction with the thirst-drive stimulus, by \tilde{s}_t . Since there are but two alternatives, it is to be expected that at the outset of training, reaction would be about 50 per cent correct. However, as the differential reinforcement yielded by the techniques employed in these investigations continues, the gradient of generalization between \tilde{s}_t and \tilde{s}_h would progressively steepen, as shown in Figure 60; i.e., discrimination learning would gradually take place, exactly as it does in fact. Thus we arrive at our twelfth corollary:

XII. *Organisms will learn to react differentially to a given objective situation according to the drive active at the time, and to react differentially to a given drive according to the objective situation at the time.*

SUMMARY

The needs of organisms operate both in the formation of habits and in their subsequent functioning, i.e., in primary motivation. Because of the sensitizing or energizing action of needs in this latter rôle, they are called *drives*.

A great mass of significant empirical evidence concerning primary motivation has become available within recent years. A survey of this material, particularly as related to hunger, thirst, injury (including the action of very intense stimuli of all kinds), sex, and the action of certain substances such as caffeine, has led to the tentative conclusion that all primary drives produce their effects by the action of various chemicals in the blood. Substances like caffeine, through bathing the neural mechanisms involved, seem to

operate by heightening the reaction potential mediated by all positive habit tendencies. Drive substances, such as the various endocrine secretions, are conceived either to be released into the blood by certain kinds of strong stimulation or as themselves initiating stimulation of resident receptors through their evocation of action by selected portions of the body, e.g., the intestinal tract and the genitalia. In both cases the energy effecting this receptor activation is called the drive stimulus (S_D).

The action of these endocrine substances, while apparently lowering the reaction threshold of certain restricted effectors (1, p. 184 ff.), seems also to have a generalized but possibly weaker tendency to facilitate action of *all* effectors, giving rise to a degree of undifferentiated motivation analogous to the Freudian libido. Thus a sex hormone would tend to motivate action based on any habit, however remote the action from that involved in actual copulation. This, together with the assumption that one or more other motivations are active to some degree, explains the continued but limited amount of habitual action of organisms when the motivation on the basis of which the habit was originally set up has presumably become zero. It also suggests a possible mechanism underlying the Freudian concept of sublimation. However, where differential behavior is required to bring about reduction in two or more drives, the differences in the drive stimuli characteristic of the motivations in question, through the principle of afferent interaction and the resulting stimulus patterning, suffice to mediate the necessary discrimination.

The hypothesis of the endocrine or chemical motivational mechanism and the associated principle of the drive stimulus, when coupled with various other postulates of the present system such as primary reinforcement, primary stimulus generalization, and the opposition of experimental extinction to excitatory potential, seem to be able to mediate the deduction, and so the explanation, of nearly all the major known phenomena of primary motivation.¹ In addition to the phenomena already summarized there may be mentioned the further deductions flowing from the system: that resistance to extinction maintains a consistent growth function of the number of reinforcements for any constant drive; that the

¹One class of phenomena seems to involve the action of fractional antecedent goal reactions and of spatial orientation. Space is not here available for the elaboration of these mechanisms and their action in motivational situations.

asymptotes of these growth functions are themselves functions of the strength of drive; that for constant habit strengths, reaction potential has a positive acceleration for increasing drives between zero and the drive employed in the original reinforcement; that if habit strength is zero, reaction tendency is zero; that an increase in drive will over-ride the total extinction of a reaction potential arising from a weaker drive; that in a given objective habit situation the abrupt shift from one drive to another will, in the absence of discriminatory training, disrupt the behavior to some extent, though not completely; that transfers of training (habits) from one motivation to another will be prompt and extensive; that organisms in the same external situations will learn to react differentially in such a way as to reduce different needs; that the conditioned evocation of endocrine secretions facilitates the evocation of muscular activity on the subsequent presentation of appropriate conditioned stimuli, which is believed to be the rôle of "emotion" in the motivation of behavior.

On the basis of the various background considerations elaborated in the preceding pages, we formulate our sixth and seventh primary molar laws of behavior:

POSTULATE 6

Associated with every drive (D) is a characteristic drive stimulus (S_D) whose intensity is an increasing monotonic function of the drive in question.

POSTULATE 7

Any effective habit strength ($s\bar{H}_R$) is sensitized into reaction potentiality (sE_R) by all primary drives active within an organism at a given time, the magnitude of this potentiality being a product obtained by multiplying an increasing function of sH_R by an increasing function of D .

From Postulates 5, 6, and 7 there may be derived the following corollary:

MAJOR COROLLARY II

The amount of reaction potentiality (sE_R) in any given potential situation is the product of (1) the effective habit strength ($s_1 + s_D\bar{H}_R$) under the existing conditions of primary drive multiplied by (2) the quotient obtained from dividing the sum of the dominant value of the primary drive (D) plus the aggregate strength of all the non-dominant primary drives (\bar{D}) active at the time, by the sum of the same non-dominant drives plus the physiological drive maximum (M_D).

NOTES

Mathematical Statement of Postulate 6

$$S_D = bf(D),$$

where

$$b > 0.$$

Mathematical Statement of Postulate 7

$$sE_R = f(s\bar{H}_R) \times f(D) \quad (33)$$

Mathematical Statement of Major Corollary II

$$sE_R = s_1 + s_D \bar{H}_R \frac{\dot{D} + D}{\dot{D} + M_D}, \quad (34)$$

where

D = the strength of the dominant primary drive at a moment under consideration

\dot{D} = the aggregate strength of all the non-dominant primary drives and quasi-drives at the moment under consideration

M_D = the physiological drive maximum (100 motes)

S_1 = the non-drive component of the stimulus complex at the moment under consideration

S_D = the stimulus specifically dependent upon the primary drive at the moment under consideration

$s_1 + s_D \bar{H}_R$ = the physiological summation of $s_1 \bar{H}_R$ and $s_D \bar{H}_R$

$s_1 \bar{H}_R$ = the effective habit loading of the non-drive component of the stimulus complex

$s_D \bar{H}_R$ = the effective habit loading of $s_D H_R$

The Equations of Perin's Graphs

The curve drawn through the upper set of data points of Figure 50 was plotted from the fitted equation:

$$n = 66(1 - 10^{-.0180 N}) - 4,$$

where n represents the number of unreinforced reaction evocations to produce experimental extinction and N represents the number of reinforcements in the setting up of the habit. The curve drawn through the lower set of data points of Figure 50 was plotted from the fitted equation:

$$n = 25(1 - 10^{-.0185 N}) - 4,$$

where n and N have the same significance as in the preceding equation. Note the practical identity of the exponents, .0180 and .0185.

The curve drawn through the data points of Figure 49 was plotted from the equation:

$$n = 9.4(10^{-.0241 N}) - 4,$$

where n means the same as above and h represents the number of hours of food privation.

The surface passing among the data points of Figure 51 was generated by the fitted equation:

$$n = 21.45 (10^{-0.00043}) (1 - 10^{-0.0182 N}) - 4 \quad (35)$$

in which n , h , and N mean the same as before. A comparison of this equation with the preceding equations shows that it is essentially the positive growth function of the first two equations in which the asymptote has been taken by a function of the drive (h) derived from the third equation. Thus n , regarded as action potentiality, may be seen to be a multiplicative function of h , or motivation, and N , or habit strength.

The Equations Employed in the Derivation of Table 5 and Figure 56

The positive growth function from which the values in column 2 of Table 5 were derived is:

$$s_1 + s_D H_R = 75(1 - 10^{-0.018 N}).$$

The equation by which the values of column 3 were derived from those in column 2 is:

$$s H_R = 100 - \sqrt{10,000 - 100 s_1 - s_D H_R}.$$

This equation is a special form of that representing the physiological summation of two habit tendencies given below.

The equation from which the values of the drive (D) were calculated from the number of hours' food privation (h) is:

$$D = \frac{100}{120} h.$$

The values of the drive deviations (d') were calculated from the equation:

$$d' = D' - D,$$

where D' represents the strength of drive employed in the formation of the habit and D represents the strength of drive under which stimulation calculated to lead to reaction evocation occurs.

The equation by means of which the values of columns 4, 5, 6, 7, and 8 of Table 5 were calculated from those of column 3 is:

$$s_D \bar{H}_R = s_D H_R (10^{-0.00831 d'}).$$

This, it may be noted, is equation 29 (p. 199 ff.), the equation of primary stimulus generalization in which $s_D \bar{H}_R$ represents the effective-habit-strength loading of the drive stimulus.

The equation by means of which the values of columns 9, 10, 11, 12, and 13 of Table 5 were calculated from those of columns 4, 5, 6, 7, and 8 is:

$$s_1 + s_D \bar{H}_R = s_1 \bar{H}_R + s_D \bar{H}_R - \frac{s_1 H_R \times s_D H_R}{100}.$$

The values of the effective drive (\bar{D}) were found by the equation:

$$\bar{D} = 100 \frac{\bar{D} + D}{\bar{D} + M_D},$$

in which \bar{D} is supposed to be the sum of the generalized effects of all the irrelevant drives active at the time, and M_D represents the maximum drive possible in a centigrade system, i.e., 100. The value of \bar{D} found by trial to fit the Perin data fairly well is 10. Therefore the equation becomes:

$$\bar{D} = 100 \frac{10 + D}{110}$$

For example, in case \bar{D} is maximal this equation becomes:

$$\begin{aligned}\bar{D} &= 100 \frac{110}{110} \\ &= 100.\end{aligned}$$

The basic equation by means of which the values of columns 14, 15, 16, 17, and 18 of Table 5 were calculated from the values of columns 9, 10, 11, 12, and 13 of Table 5, is:

$$sE_R = s_1 + s_D \bar{H}_R \times \frac{\bar{D}}{100}.$$

Substituting the equivalent of \bar{D} and simplifying, this becomes:

$$sE_R = s_1 + s_D \bar{H}_R \times \frac{10 + D}{110}.$$

REFERENCES

1. BEACH, F. A. Arousal, maintenance, and manifestation of sexual excitement in male animals. *Psychosomatic Medicine*, 1942, 4, 173-198.
2. ELLIOTT, M. H. The effect of change of drive on maze performance. *Calif. Pub. in Psychol.*, 1929, 4, 185-188.
3. FINCH, G. Hunger as a determinant of conditional and unconditional salivary response magnitude. *Amer. J. Physiol.*, 1938, 123, 379-382.
4. HEATHERS, G. L., and ARAKELIAN, P. The relation between strength of drive and rate of extinction of a bar-pressing reaction in the rat. *J. Gen. Psychol.*, 1941, 24, 243-258.
5. HOVLAND, C. I. The generalization of conditioned responses: II. The sensory generalization of conditioned responses with varying intensities of tone. *J. Genet. Psychol.*, 1937, 51, 279-291.
6. HULL, C. L. Differential habituation to internal stimuli in the albino rat. *J. Comp. Psychol.*, 1933, 16, 255-273.
7. HULL, C. L. The rat's speed-of-locomotion gradient in the approach to food. *J. Comp. Psychol.*, 1934, 17, 393-422.
8. LEEPER, R. The role of motivation in learning: A study of the phenomenon of differential motivational control of the utilization of habits. *J. Genet. Psychol.*, 1935, 46, 3-40.
9. MILLER, N. E., and MILES, W. R. Effect of caffeine on the running speed of hungry, satiated, and frustrated rats. *J. Comp. Psychol.*, 1935, 20, 397-412.
10. MOORE, C. R., PRICE, D., and GALLAGHER, T. F. Rat prostate cystology and testes-hormone indicator and the prevention of castration changes by testes extract injection. *Amer. J. Anat.*, 1930, 45, 71-108.
11. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.

12. PERIN, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. *J. Exper. Psychol.*, 1942, *35*, 93-113.
13. SKINNER, B. F. *The behavior of organisms*. New York: D. Appleton-Century Co., Inc., 1938.
14. SKINNER, B. F., and HERON, W. T. Effect of caffeine and benzedrine upon conditioning and extinction. *Psychol. Record*, 1937, *1*, 340-346.
15. STONE, C. P. The retention of copulatory ability in male rats after castration. *J. Comp. Psychol.*, 1927, *7*, 369-387.
16. STONE, C. P. The retention of copulatory activity in male rabbits following castration. *J. Genet. Psychol.*, 1932, *40*, 296-305.
17. STONE, C. P. Activation of impotent male rats by injections of testosterone propionate. *J. Comp. Psychol.*, 1938, *25*, 445-450.
18. SWITZER, S. A. The effect of caffeine on experimental extinction of conditioned reactions. *J. Gen. Psychol.*, 1935, *12*, 78-94.
19. WARDEN, C. J., JENKINS, T. N., and WARNER, L. H. *Introduction to comparative psychology*. New York: Ronald, 1934.
20. WILLIAMS, S. B. Resistance to extinction as a function of the number of reinforcements. *J. Exper. Psychol.*, 1938, *23*, 506-521.
21. YOUNG, P. T. *Motivation of behavior*. New York: John Wiley and Sons, 1936.
22. ZENER, K. E., and MCCURDY, H. G. Analysis of motivational factors in conditioned behavior: I. The differential effect of changes in hunger upon conditioned, unconditioned, and spontaneous salivary secretion. *J. Psychol.*, 1939, *8*, 321-350.

CHAPTER XV

Unadaptive Habits and Experimental Extinction

Science tacitly assumes that similar causes will be followed by similar effects. In the field of behavior dynamics it is accordingly to be expected that an act which has been followed by a need reduction in a given situation will always be so reinforced whenever the reaction occurs in other exactly similar situations. It may be noted in this connection first that, as a rule, most of the factors of such situations possess energies which activate one or more of the receptors of the reacting organism. Secondly, according to the law of reinforcement (p. 80 ff.), all stimuli whose receptor discharges are contiguous with reactions which are followed by reinforcing states of affairs tend to acquire the capacity of later evoking that reaction. As a result of this combination of circumstances it comes about that on the recurrence of the situation in question (including the need) the corresponding stimuli must also recur, they will evoke the reaction conditioned to them, the need will be reduced, and survival will be facilitated.

At this point of the analysis, however, serious complications appear. In the first place, exact duplicates of situations probably never recur. A second complication is that by no means all of the factors of any reaction situation are critical in the sense that their presence is necessary for the act in question to bring about need reduction. A third and closely related complication lies in the fact that the really critical factor or factors of the reaction situation may not stimulate the receptors of the reacting organism at all; in the field of vision, for example, the view of the critical factor may be cut off by the interposition of a completely irrelevant object. Since organisms have no inner monitor or entelechy to tell them in advance which stimulus elements or aggregates are associated with the critical causal factor or factors of reaction situations, *the law of reinforcement, other things equal, will mediate the connections of the non-critical stimulus elements to the reaction quite as readily as those of the critical ones.*

As a result of the largely random flux of events in the world to which organisms must react, it inevitably comes about that they will often be stimulated by extensive groups of conditioned stim-

ulus elements, *none* of which is causally related to the critical factor or factors in the reinforcement situation. In such cases, if the stimuli evoke the reaction it will not be followed by reinforcement. This, of course, is wasteful of energy and therefore unadaptive. The necessarily unadaptive nature of an appreciable portion of the habits set up by virtue of the law of reinforcement naturally raises the question of how organisms are able to survive under such conditions (7, p. 501). The answer is found in the behavioral principle known as *experimental extinction*. This is the subject of the present chapter.

CONCRETE EXAMPLES OF EXPERIMENTAL EXTINCTION

The principle of experimental extinction is so ubiquitous a factor in behavior dynamics that it can hardly escape anyone's observation. The dog which has been taught to "speak" for food soon ceases to do this if the food, petting, etc., is systematically withheld following the act. The principle has even passed into folklore, as shown by the fable of the boy who shouted "Wolf! Wolf!" when no wolf was near. After a few such false alarms the rescuing behavior of the hearers would inevitably become extinguished, and they would cease to respond to the calls quite as the fable states.

The systematic investigation of experimental extinction originated in Pavlov's conditioned-reflex laboratory in Petrograd. Since the comparative simplicity of the conditioned-reflex technique brings out with maximum clarity the essential principles involved, a description of one of Pavlov's experiments will serve as a useful introduction to the technical aspects of the subject, even though the artificiality of the experiment may tend somewhat to obscure the functional significance of the principle. Pavlov reports having produced a conditioned reflex by first showing a dog some meat powder and then letting him eat it. After a considerable number of reinforcements extending over several days, the mere visual stimulation of the food would evoke a profuse flow of saliva. The meat powder was then presented at a distance for a number of 30-second periods, but without being followed by the customary feeding. On each of the latter occasions the number of cubic centimeters of saliva secreted was recorded. The results of this procedure are shown in Table 6, where it may be seen (1) that after only a few non-reinforced reactions the visual stimulus completely loses its

TABLE 6

TABLE SUMMARIZING THE RESULTS OF AN EXPERIMENT INVOLVING EXPERIMENTAL EXTINCTION, PERFORMED IN PAVLOV'S LABORATORY (9, p. 58).

Successive Unreinforced Stimulations	Number of Cubic Centimeters of Saliva Secreted in Each 30-Second Period
1	1.0
2	.6
3	.3
4	.1
5	.0
6	.0

power of reaction evocation, and (2) that the course of this loss is progressive, the rate of fall being more rapid at first than later.

EXPERIMENTAL EXTINCTION AS A FUNCTION OF THE NUMBER OF UNREINFORCED REACTIONS

The process of experimental extinction has now been studied in many other laboratories where many different reactions have been extinguished under many different conditions. One of the more easily interpreted of these studies has been reported by Holland (6). This investigator conditioned a simple sinusoidal sound wave of 1000 cycles per second to the galvanic skin reaction in 20 human subjects; the 24 reinforcements (a weak electric shock to the wrist) by which the habit was originally set up were separated by 30-minute rest pauses after the first and second series of eight. The habit was then extinguished by repeated evocations of the act without reinforcement. The pooled results of the first five reactions of this extinction process presumably yield a set of values closely approximating the typical rate of extinction. They are represented graphically by the circles of Figure 57. In order to determine more precisely the characteristics of this negative learning curve, a simple negative growth function was fitted to the values represented by the circles. From this the smooth curve passing among the circles was plotted. A glance at this curve shows that, except at the zero point, the fit is excellent. The failure of the amplitude of the reaction at point 0 to be as high, relatively, as the other amplitudes may plausibly be interpreted as due to "inhibition of reinforcement" (see p. 289 ff.), which is subsequently

"disinhibited" in part, at least, by the abrupt change in experimental routine incidental to the process of extinction (5). We conclude, then, that the curve of experimental extinction when uncomplicated by irrelevant factors is probably a simple negative growth function.

Further examination of the smooth curve in Figure 57 reveals two additional characteristics which merit consideration. The first

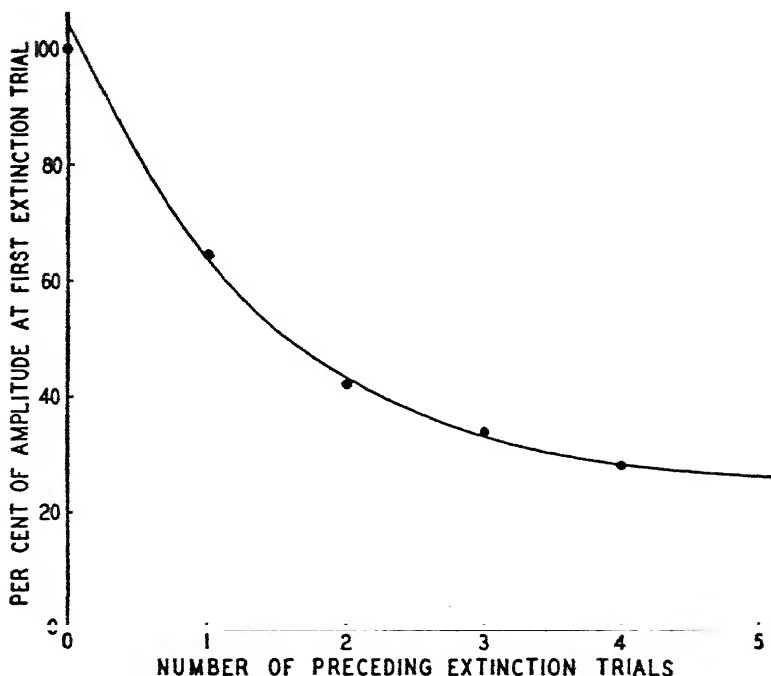


FIG. 57. A graphic representation of the course of habit decrement as a function of successive unreinforced evocations of the previously learned act. (The data were received from Hovland in a private communication.)

is that the asymptote (limit of fall) of the curve is not zero, but about 24 per cent of the amplitude of what the reaction was before extinction began. This is probably an artifact due to the well-known tendency of the skin to yield appreciable galvanic reactions even to mild stimulations previous to any specific conditioning whatever (see p. 186 ff.). In this respect it is believed that the Pavlovian results shown in Table 6 more truly represent the quantitative principle of experimental extinction. The second notable

characteristic of the function shown in Figure 57 is its strikingly rapid rate of decrement, its F -value being a little less than $\frac{1}{2}$. By way of contrast we may consider the rate of acquisition of a comparable habit, which is shown above in Figure 21 (p. 103). The fractional increment (F) of this latter growth function is approximately $1/14$, which corresponds to a radically slower rate of change. Thus we arrive at the indication that the rate of decrement under the present set of conditions is more rapid than is the rate of the original acquisition of the habit.

In this connection it must be pointed out that of those reactions extensively investigated, the galvanic skin reaction and the salivary reaction both show a progressive decrement in amplitude as experimental extinction progressively reduces the excitatory tendency to zero. The typical motor reaction differs sharply from this by displaying under comparable conditions mainly an increase in latency and a decrease in *probability of occurrence*. Motor reactions under certain conditions, at least, are apt to show an increase in reaction intensity in the early stages of extinction, though in the later stages there is usually a slight tendency to a diminution in the intensity of the reaction (2, p. 148). Because of these differences it is probable that the salivary reaction is the one best adapted for the quantitative determination of the characteristics of the curves of both simple learning and simple extinction.

THE STIMULUS GENERALIZATION OF EXTINCTION EFFECTS

In an earlier chapter (p. 183 ff.) we saw that habits manifest the phenomenon of stimulus generalization. Since physiologically maladaptive habits (when first formed) are no different than other excitatory tendencies, it is to be expected that they likewise will generalize. We have also seen that experimental extinction purports to be a kind of corrective mechanism. It is evident, however, that if experimental extinction is to correct false habit tendencies with reasonable efficiency, extinction effects must also generalize; if extinction were a mere point phenomenon, false receptor-effector connections would probably never become wholly eradicated because the process of extinguishing a zone consisting of infinitesimal points would be literally interminable.

As a matter of fact, extinction effects do manifest stimulus generalization to a marked degree. This important phenomenon,

like so many others in this field, also appears first to have been investigated in Pavlov's laboratory. For example, the sound of a buzzer, the sound of a metronome, and a tactile stimulation were separately conditioned in a dog to the salivary reaction induced by having weak acid injected into his mouth. At the conclusion of the positive training the buzzer evoked 13 drops per 30-second period, the metronome, 12 drops, and the tactile stimulus, 4 drops. The metronome was then extinguished by unreinforced presentations at 3-minute intervals until it evoked no secretion whatever. A few minutes later the secretion evoked by the tactile stimulus had fallen to zero and that by the buzzer had fallen to 2.5 drops (9, p. 55). The effects of the extinction of the metronome habit had clearly generalized in such a way as to inhibit completely the tactile habit and almost to inhibit the buzzer habit.

Pavlov even investigated the quantitative gradient of generalized extinction effects. He set up a number of "homogeneous" conditioned reflexes to a series of points on the skin, then extinguished the reaction whose stimulus was located at one extreme of the series and noted the extent to which the other conditioned reflexes were weakened as a result. On the basis of such experiments, Pavlov concludes (9, p. 158):

It is plain that, the further away on the skin the secondarily inhibited place is from the place which undergoes the primary inhibition [extinction], the weaker is the irradiated inhibitory after-effect.

Subsequently, Anrep (1), Bass and Hull (3), and Hovland (6) sought to plot the generalization gradient of extinction effects with progressively more refined experimental procedures. Anrep employed cutaneous stimulation with the salivary reaction to food in dogs; Bass and Hull employed cutaneous stimulation with the galvanic skin reaction evoked by an electric shock in humans.

Hovland conditioned the pitch of four pure tones an equal amount to the galvanic skin reaction evoked originally by a weak electric shock. The tones were so chosen that they differed from each other by 25 discrimination thresholds (j.n.d.'s). Then a tone at one or the other extreme of the series was extinguished to a partial but known degree, after which all four tones were tested to determine the strength of the residual excitatory tendency evocable by each. The pooled results of the twenty subjects employed in this investigation are represented graphically by the circles in

Figure 58. This series of circles constitutes a clear verification of the gradient of generalized extinction effects reported by Pavlov. It is evident that experimental extinction is an extended and not a point phenomenon.

The precision and general reliability of Hovland's data also warranted an attempt at a determination of the mathematical

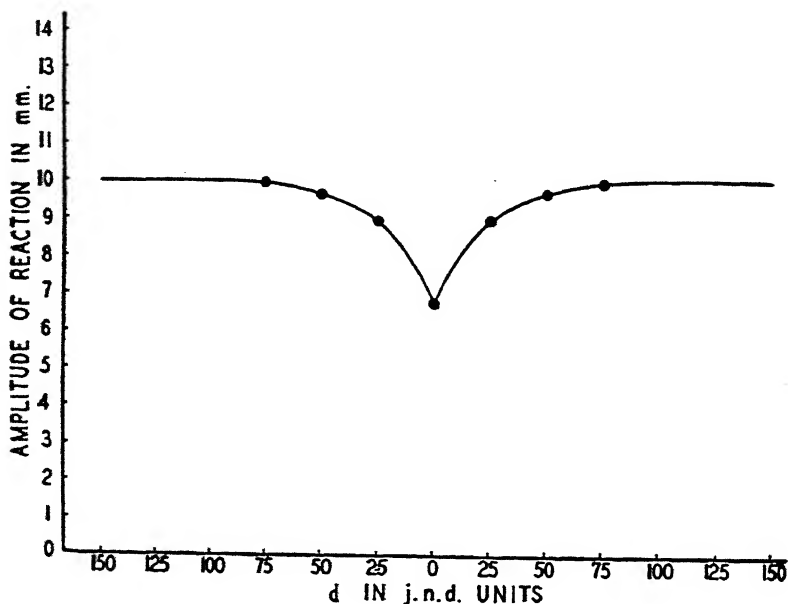


Fig. 58. Empirical stimulus generalization gradient of an extinguished galvanic skin reaction plotted from data published by Hovland (6). The gradient extends in both directions on the stimulus continuum (rate of vibration) from the point extinguished (0); d is the difference between the stimulus originally conditioned and the stimulus evoking the reaction. Note that here the gradients are directly the reverse of those shown in the closely related Figure 42, p. 185.

characteristics of the gradient. To this end, an equation was fitted to the values represented by the circles. From this equation was plotted the smooth curve running among them. That the equation, a simple positive growth function, fits the data rather well may be seen by an inspection of the figure. We accordingly conclude that the gradient of stimulus generalization extinction effects is probably a simple positive growth function.

THE INTERACTION OF THE GRADIENTS OF EXCITATION AND OF EXTINCTION

It is clear from a comparison of Hovland's excitation and extinction gradients (Figures 58 and 42, p. 264 and 185), that if their parameters turn out to be alike the second is exactly the shape which would be required completely to eliminate the first. The conditions of the two experiments are such that an exact agreement is not to be expected between the two maximum opposing values. In the matter of the constant incremental factor of change

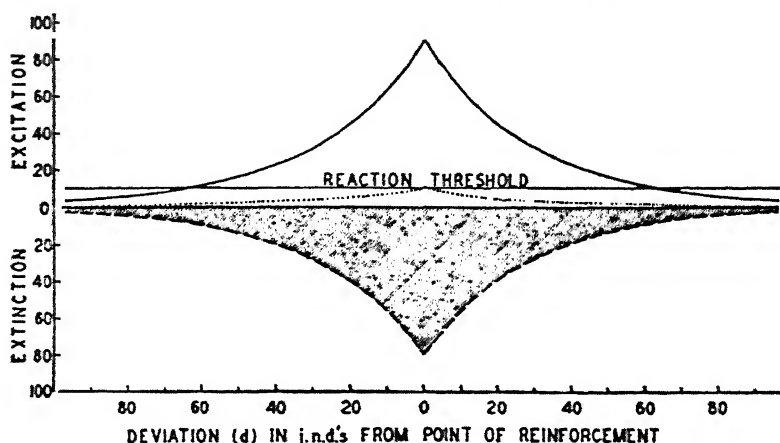


FIG. 59. Diagram representing the manner in which the gradients of experimental extinction are supposed, theoretically, to interact with the gradients of a false or unadaptive reaction tendency in such a way as to eliminate the latter. The excitatory gradients are represented by the upper curves, those of extinction effects are represented by the lower curves, and the residue of effective excitatory tendency is represented by the dotted line in between.

(F), which might easily be the same, we find that agreement also does not exist; the F -factor of the excitation generalization is approximately $1/33$, whereas that of extinction is approximately $1/21$, the range of the former being appreciably the greater. A difference of this order might, however, easily arise from sampling "errors." A considerable amount of careful quantitative research is badly needed to clear up this matter.

The manner in which the two gradients are conceived to interact in the elimination of the unadaptive effects of false (and therefore unadaptive) reaction tendencies is shown in Figure 59. In this figure the upper pair of gradients represent excitation. They

are drawn on the assumption that reinforcement occurred at point zero on the stimulus continuum and diminished at the rate of approximately $1/29$ at each additional j.n.d. of deviation of the evoking stimulus. This value was chosen because it falls about midway between the F -values of Hovland's two gradients. Extinction is assumed also to have taken place at zero on the stimulus continuum, and to have continued until no reaction would be evoked by that stimulus, i.e., until the reaction tendency had passed beneath the reaction threshold. Since the reaction threshold is taken at 10 units, this means that the extinction effects must have become great enough to neutralize $90 - 10$, or 80 points of excitation; i.e., the extinction effects must possess a *negative* strength of 80 units. The generalization gradients of these extinction effects are plotted on the basis of the same F -constant as are those of the excitation effects, viz., $1/29$. By subtracting the extinction gradient from the corresponding excitation gradient, we obtain the effective or residual strength of the excitatory tendency, which is represented by the dotted line. This shows that at all points on the stimulus continuum the effective excitatory tendency is below the reaction threshold, i.e., at no point on the stimulus continuum will the unadaptive reaction be evoked.

A rather different picture, and one of great theoretical significance, emerges when extinction takes place, not at the point of reinforcement but out on one of the wings of the excitation gradient (8, p. 25). In nature this typically occurs when generalization has extended on the stimulus continuum to a point where the reaction will no longer be reinforced. Such a situation calls for *discrimination* on the part of the organism; thus primitive stimulus generalization is in a sense indiscriminate and is the natural antithesis of discrimination. For example, in Figure 59 the excitatory tendency before extinction would evoke reaction with varying degrees of intensity or probability anywhere on the stimulus continuum within about 64 j.n.d.'s of the point originally reinforced.

Suppose, however, that the stimulus at 8 j.n.d.'s on one side of the point of reinforcement represents a state of affairs which in conjunction with the habituated act will not yield reinforcement. Suppose further that this stimulus is presented to the subject repeatedly until it will no longer evoke the reaction to any degree whatever. The interaction of the generalization gradients of the extinction effects so produced, with those of the original excitation gradients, is shown in Figure 60. There, as in Figure 59, it may

be seen by the course of the dotted line that the point extinguished diminishes the effective excitatory tendency to the reaction threshold, a reduction of some 58 points. It will also be noticed that at no place beyond this point does the effective excitatory tendency rise above the reaction threshold. However, between the point extinguished and the point of original reinforcement the curve of effective excitation rises steeply to a value of 45, which is far above the reaction threshold. This means that through the interaction

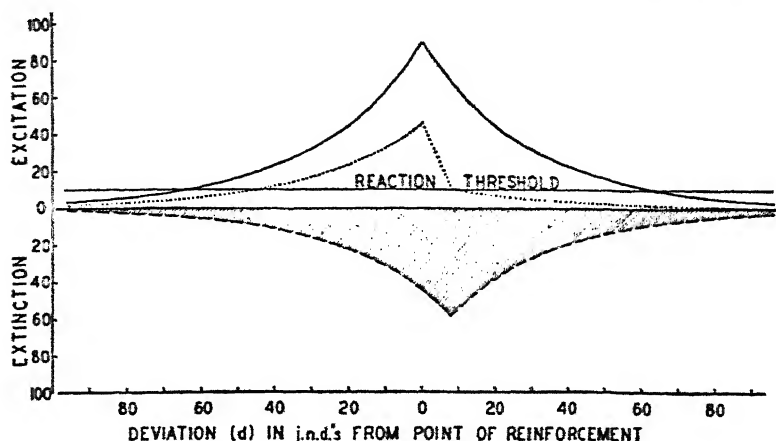


FIG. 60. Diagram representing the manner in which the gradients of experimental extinction are supposed, theoretically, to interact with the gradients of a positive reaction tendency to generate the phenomenon of discrimination learning. As in Figure 59, the upper curve represents excitation, the lower curves represent extinction effects, and the dotted line in between represents the residual effective reaction tendency. Note the greatly steepened gradient on the latter curve between 0 and 8 j.n.d.'s. It is largely because of this that the improvement in simple discrimination is believed to occur.

of the gradients of excitation and extinction the phenomenon of simple discrimination learning has been generated as a secondary principle.¹

THE REACTION GENERALIZATION OF EXTINCTION EFFECTS

Just as the adaptive aspects of behavior dynamics require a stimulus generalization of extinction effects to neutralize effectively

¹This view of simple discrimination learning follows substantially the approach developed by Spence in numerous theoretical and experimental studies (10, 11, 12, 13, 14).

the unadaptive habits inevitably set up in considerable numbers by the indiscriminate action of the law of reinforcement, so the reaction generalization tendencies of unadaptive habits require for the survival of organisms that there be a complementary reaction generalization tendency of extinction effects. Here again Pavlov made the initial discovery. He reports (9, p. 54):

This latter phenomenon [generalized extinction effects] involves not only those conditioned reflexes which were based upon a common unconditioned reflex with the primarily extinguished one (*homogeneous conditioned reflexes*), but also those which were based upon a different unconditioned reflex (*heterogeneous conditioned reflexes*).

In Pavlov's terminology, if a tone and a tactile stimulus were each separately conditioned to an alimentary salivary secretion by the use of food reinforcement, the resulting reflexes would be *homo-*

geneous. If, on the other hand, the tone were reinforced with food and the tactile stimulus were reinforced by weak acid being injected into the mouth, the tactile conditioned reaction would be defensive and observably different in nature; the latter two conditioned reactions would accordingly be called *heterogeneous*.

An analogous phenomenon in a selective learning situation was reported by Youtz (15); a closely related study worked out with meticulous care has been performed by Ellson (4). In the latter investigation albino rats were placed, one at a time, in a small, sound-

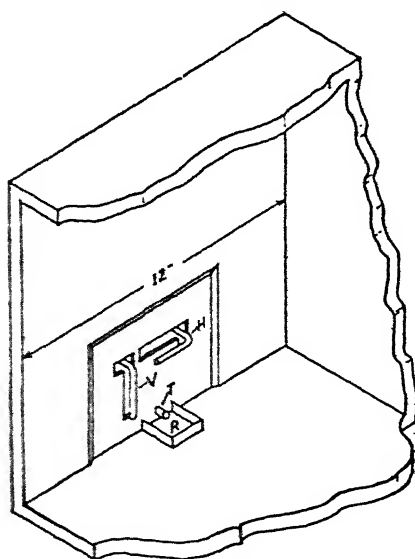


FIG. 61. Sketch showing the two manipulation bars employed in Ellson's experiment (4, p. 341).

shielded, cubical space. From one wall of the chamber there projected two bars, as shown in Figure 61. These bars could be introduced into the chamber or retracted at will, without opening the chamber door. During the learning and extinction processes

only one bar was presented at a time. A lump of moist food was placed behind the panel so that its odor would be carried through the bar slot by the ventilating system. By investigating this odor the animal sooner or later would move the bar in question, the horizontal bar downward, and the vertical bar to the left. Either movement caused a magnetic release mechanism behind the panel to give a click and at the same time a cylinder of food dropped into a cup beneath. In this way the horizontal-bar habit was set up; the vertical-bar habit was set up 30 minutes later in an exactly analogous manner.

On the next day the two habits were extinguished in succession, first the vertical-bar habit, and 5.5 minutes later the horizontal-bar habit; this was done by severing the electrical connection between the manipulative bars and the food-release mechanism. The horizontal-bar habit was extinguished in control animals without a preceding extinction on the vertical-bar habit. It was found that on the average the control animals operated the horizontal bar 49.7 times before a given degree of extinction supervened, whereas 5.5 minutes after the extinction of the vertical-bar habit the experimental animals extinguished on the horizontal bar to the same degree after only 23.9 operations of the bar. This reduction in resistance to experimental extinction of approximately 50 per cent clearly suggests reaction generalization of extinction effects.

It is to be noted, incidentally, that neither in the Pavlovian experiments nor in that of Ellson is the reaction generalization uncomplicated by stimulus generalization. In both cases there was a decided element of stimulus similarity between the original extinction and the subsequent generalization-of-extinction situation in that the incidental stimuli from the apparatus environment were identical. Nevertheless, these experiments demonstrate that extinction effects are transferable from one reaction to another reaction which is to a considerable extent different.

THE SPONTANEOUS RECOVERY OF EXTINCTION EFFECTS

The account of experimental extinction contained in the preceding sections of the present chapter, together with the somewhat misleading use of the word *extinction* in this connection, might easily suggest to the uninitiated that experimental extinction necessarily abolishes completely and permanently the unadaptive reaction tendency involved. This is far from being the case, as Pavlov

himself long ago pointed out (4, p. 60). That a reaction tendency may be very much alive after total experimental extinction is demonstrated in a striking manner by the fact that if the conditioned stimulus is withheld from the organism for some time after experimental extinction has occurred, its reapplication will evoke the reaction to a considerably greater extent than it did at the conclusion of the original extinction. This is known as *spontaneous recovery*.

The phenomenon of spontaneous recovery was discovered by Pavlov; there is accordingly a certain appropriateness in choosing our initial illustration of it from his writings. This example comes as a sequel to an extinction experiment reported above (p. 259 ff.) and summarized in Table 6. Following the extinction there described, the dog was left to itself for two hours, after which the conditioned stimulus (visual presentation of meat powder) was again delivered. This was followed by .15 cubic centimeters of salivary secretion, which showed that the reaction tendency had spontaneously recovered about one-sixth of its original strength. Usually the rate of recovery is greater than this.

Proceeding now to the consideration of the quantitative law of the spontaneous recovery of experimental extinction as a function of time, we turn to another portion of the Ellson experiment described in the preceding section of the present chapter (p. 268 ff.). Four groups of 25 albino rats were trained by means of an equal number of food reinforcements to depress a horizontal bar (Figure 61) until the habit was thoroughly learned. They were then presented with the bar and permitted to operate it without reinforcement until a period of five minutes elapsed without recorded pressures. Following this one group was extinguished to the same degree a *second* time after a recovery period of 5.5 minutes, another group was again extinguished after 25 minutes, a third group after 65 minutes, and a fourth group after 185 minutes. The solid circles in Figure 62 show the median number of unreinforced reactions required again to produce extinction after the several recovery periods. It is evident at a glance that spontaneous recovery is considerable in amount, and that it is an increasing function of the duration of the recovery period.

In an attempt to determine this relationship more precisely, a positive growth function was fitted to these values; this is represented by the smooth curve drawn among the solid circles. While the fit is by no means perfect, it is evident that the spontaneous

recovery from primary experimental extinction effects is approximately a simple positive growth function. An inspection of this curve shows that at 185 minutes it practically reaches its asymptote, or limit of rise. It is also to be noted that *this maximum is only about 50 per cent of the original strength of the habit*, which is indicated by the broken line at the top of the figure.

We saw in the last section (p. 267 ff.) that extinction effects manifest reaction generalization, which raises the question of the

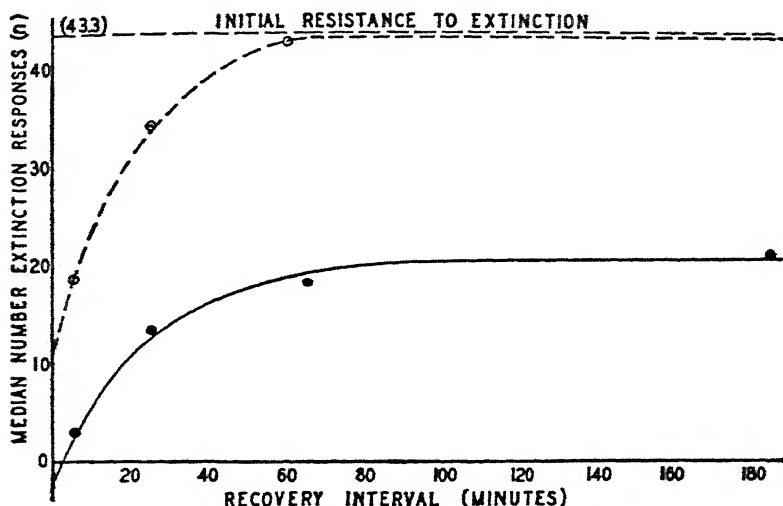


FIG. 62. Graphic representation of Ellson's empirical values for the spontaneous recovery of a habit from primary experimental extinction (lower curve, solid circles) and from reaction-generalized experimental extinction effects (upper curve, hollow circles). Both curves represent simple positive growth functions fitted to the circles through which they pass. (Plotted from data published by Ellson, 4.)

quantitative law regarding the spontaneous recovery of the reaction generalization of extinction effects. This problem also was investigated by Ellson as a portion of the experiment just described. Four additional groups of animals were extinguished on the vertical-bar habit, and then after recovery periods of 5.5, 25, 65, and 185 minutes they were extinguished on the horizontal-bar habit. The median number of unreinforced reactions required to produce experimental extinction are shown by the hollow circles of Figure 62.

A simple growth function was fitted to these values; this is

represented by the smooth, broken-line curve drawn among the circles. Here again the fit is not very close, though there is an indication that spontaneous recovery from reaction-generalized extinction effects approximates a simple growth function. It is to be noted that this curve also approximately reaches its limit of rise at 185 minutes, which, incidentally, almost exactly equals the number of unreinforced reactions required to extinguish the horizontal bar when this extinction is *not* preceded by the extinction of the vertical-bar habit. Moreover, it is probably significant that the rate of rise of this curve is very close to that of the one fitted to the data derived from the spontaneous recovery of primary extinction effects; the fractional rate of change (F) of the latter is $1/21$, and that of the former is approximately $1/24$.

THE DISINHIBITION OF EXTINCTION EFFECTS

A second phenomenon which demonstrates that experimental extinction to the point of zero reaction does not necessarily abolish a reaction tendency permanently and completely is that known as *disinhibition*. This was pointed out by Pavlov, in whose laboratory the phenomenon was originally discovered. The nature of disinhibition is nicely illustrated by the following account.

Dr. Zavadsky, one of Pavlov's pupils, experimented with a dog which had two salivary fistulas, one from the submaxillary and the other from the parotid gland. Through repeated presentations and ingestions, the sight and odor of meat powder presented at a distance had become conditioned to evoke the salivary reaction of both glands. Thereupon occurred the events summarized in Table 7, where it may be seen that the first three stimulations were not reinforced. An extremely rapid experimental extinction resulted, shown by the fact that at the third stimulation the reaction was zero. On the fourth trial, however, the presentation of the meat powder was accompanied by an "extra" stimulus in the form of a cutaneous vibration. In this case three drops of saliva were secreted, which indicates that the inhibition was partly abolished (disinhibited). Five minutes later when the meat powder was presented it was accompanied by knocks under the table; disinhibition was again manifested by a secretion of two drops. After five minutes the meat powder was presented alone; the zero reaction to this indicates that the extinctive inhibition had returned, following the preceding disinhibition. This observation illustrates

TABLE 7

SUMMARY OF DR. ZAVADSKY'S EXPERIMENTAL RESULTS ILLUSTRATING BOTH SIMULTANEOUS AND PERSEVERATIVE ASPECTS OF DISINHIBITION (9, p. 65).*

Time of Occurrence	Stimulus Applied During One Minute	Amount of Saliva in Drops During One Minute	
		From Submaxillary Gland	From Parotid Gland
1:53 P.M.	Meat powder presented at a distance	11	
1:58 P.M.	Meat powder presented at a distance	4	
2:3 P.M.	Meat powder presented at a distance	0	
2:8 P.M.	Same + tactile stimulation of skin...	3	
2:13 P.M.	Same + knocks under the table.....	2	
2:18 P.M.	Meat powder at a distance.....	0	
2:20 P.M.	Prof. Pavlov enters the room containing the dog, talks, and stays for two minutes.....		
2:23 P.M.	Meat powder at a distance.....		2
2:28 P.M.	Same.....		0

* Previous to this experiment it had been shown repeatedly that neither the tactile nor the auditory stimulus, nor the entry of Professor Pavlov into the experimental room, produced any secretory effect at all.

the transitory nature of disinhibition, which presumably is due to the fading out of the stimulus trace of the extra stimulus. It is to be noted (Table 7) that no disinhibition was followed by a reaction nearly as great as the eleven drops evoked at 1:53 P.M. preceding the experimental extinction.

The reactions just considered are cases of *simultaneous* disinhibition. In spite of its clearly transitory nature, disinhibition shows a certain tendency to perseveration, or after-effect. The entrance of Professor Pavlov into the experimental room for two minutes served as an obvious external stimulation. One minute after he had left the meat powder was presented at a distance and it evoked the conditioned reaction of five drops; this illustrates the *perseverative* effect of disinhibition. Five minutes later, however, the same stimulus produced no reaction, which shows that the perseveration was distinctly brief in duration.

SUMMARY

The conditions under which reinforcements occur inevitably set up many receptor connections that are false in the sense that if the stimulus elements in question should alone evoke the reaction,

reinforcement would not follow. The functional corrective of this unadaptive aspect of the law of reinforcement is experimental extinction. This consists of a progressive weakening of the reaction tendency whenever the evocation of the reaction is not followed by adequate reinforcement. Experiments suggest that the reaction tendency diminishes as a negative growth function of the number of closely successive unreinforced evocations. In one study in which analysis was made of the relevant learning curves, it was found that the rate of loss through extinction was much faster than the rate of the original acquisition of the reaction tendency.

Just as positive habits manifest stimulus generalization, so do extinction effects also manifest this tendency. Moreover, the gradient of the generalization of extinction effects is of such a nature that if the incremental factor (F) in the two cases were the same the extinction of a habit at the point of reinforcement on the stimulus continuum would completely neutralize the reaction tendency, not only at that point but at all other points on the stimulus continuum to which it would show primary stimulus generalization.

In case experimental extinction occurs on one wing of a positive generalization gradient, the interaction of this with the resulting extinction generalization gradient produces a greatly steepened gradient of that portion of the effective reaction tendency lying between the point of extinction and the point of the original reinforcement. This steepened gradient leaves the latter stimulus still able to evoke the reaction, while the former does not. The result is a distinctly heightened power of discrimination.

In both conditioned-reflex and selective-learning situations, extinction effects manifest reaction generalization, quite as do positive habit tendencies.

Experimental extinction does not necessarily abolish completely and permanently the reaction tendency extinguished; this is shown by the phenomenon of spontaneous recovery. Spontaneous recovery, of both primary extinction and response-generalization extinction effects, takes place approximately as a positive growth function of time elapsing since the termination of the extinction process. Under the continuous action of a static conditioned stimulus, spontaneous recovery of both primary and generalized extinction nearly reaches its maximum at three hours. Maximal spontaneous recovery of primary extinction effects under these conditions is about

50 per cent, whereas that of generalized extinction effects is approximately 100 per cent.

A second phenomenon, known as disinhibition, further demonstrates that experimental extinction does not necessarily constitute an abolition of the extinguished reaction tendency. This means that a weak "extra" stimulus will partially restore an extinguished reaction tendency. Such restorations are quite transitory, however. Disinhibition may partially restore an extinguished reaction tendency even when the extra stimulus has been delivered several minutes before the attempt is made to evoke the extinguished reaction.

NOTES

The Equation Fitted to Hovland's Curve of Extinction Data Following Distributed Practice

The equation from which was plotted the smooth curve of Figure 57 is:

$$A = 24.1 + 80.9 \times 10^{-.315 N},$$

where A is the amplitude of the galvanic skin reaction evoked by the conditioned stimulus, and N is the number of the preceding extinction repetitions. In this equation the exponential value of .315 corresponds to a factor of reduction (F) of 1/1.94.

The Equation Fitted to Hovland's Generalized Extinction Data

The equation from which the smooth curve of Figure 58 was plotted is:

$$A = 6.7 + 3.25(1 - 10^{-.0195 d}),$$

where A is the amplitude of the galvanic skin reaction evoked by a stimulus, and d is the difference in j.n.d.'s between that stimulus and the one to which the reaction was originally extinguished.

The comparable equation fitted to the same author's published empirical data on *excitatory* generalization effects is:

$$A = 12.6 + 6 \times 10^{-.0135 d}.$$

The F -value corresponding to .0135 is approximately 1/33; that corresponding to .0195 is approximately 1/21. Unfortunately it is not known whether either of these F -values is constant under all conditions, and if not, upon what their magnitude may depend.

The Equations of the Curves Shown in Figure 62

The equation of the curve of the spontaneous recovery of primary extinction effects shown in the lower portion of Figure 62 is:

$$n = 22.2 (1 - 10^{-.018 t'''}) - 2,$$

where n is the number of unreinforced reactions required to produce the second experimental extinction, and t''' is the time in minutes of the recovery period.

i.e., from the termination of the first experimental extinction to the beginning of the second experimental extinction.

The equation of the curve of spontaneous recovery from the reaction generalization of experimental extinction is:

$$n = 43 - 32 \times 10^{-.021 t'''},$$

where n has the same significance as in the preceding equation, and t''' is the time in minutes of the recovery period, i.e., from the termination of the extinction of the vertical-bar habit to the beginning of the extinction of the horizontal-bar habit.

The rate of rise to its asymptote of the curve represented by the first equation (F) is approximately $1/24$; that of the second is $1/21$.

REFERENCES

1. ANREP, G. V. The irradiation of conditioned reflexes. *Proc. Royal Soc. London*, 1923, 94, Series B, 404, 425.
2. ARAKELIAN, P. Cyclic oscillations in the extinction behavior of rats. *J. Gen. Psychol.*, 1939, 21, 137-162.
3. BASS, M. J., and HULL, C. L. The irradiation of a tactile conditioned reflex in man. *J. Comp. Psychol.*, 1934, 17, 47-65.
4. ELLSON, D. G. Quantitative studies of the interaction of simple habits. I. Recovery from specific and generalized effects of extinction. *J. Exper. Psychol.*, 1938, 23, 339-358.
5. HOVLAND, C. I. 'Inhibition of reinforcement' and phenomena of experimental extinction. *Proc. Natl. Acad. Sci.*, 1936, 22, 430-433.
6. HOVLAND, C. I. The generalization of conditioned responses: I. The sensory generalization of conditioned responses with varying frequencies of tone. *J. Gen. Psychol.*, 1937, 17, 125-148.
7. HULL, C. L. A functional interpretation of the conditioned reflex. *Psychol. Rev.*, 1929, 36, 498-511.
8. HULL, C. L. The problem of stimulus equivalence in behavior theory. *Psychol. Rev.*, 1939, 46, 9-30.
9. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
10. SPENCE, K. W. The nature of discrimination learning in animals. *Psychol. Rev.*, 1936, 43, 427-449.
11. SPENCE, K. W. The differential response in animals to stimuli varying within a single dimension. *Psychol. Rev.*, 1937, 44, 430-444.
12. SPENCE, K. W. Analysis of formation of visual discrimination habits in chimpanzee. *J. Comp. Psychol.*, 1937, 23, 77-100.
13. SPENCE, K. W. Failure of transposition in size discrimination of chimpanzees. *Amer. J. Psychol.*, 1941, 54, 223-229.
14. SPENCE, K. W. The basis of solution by chimpanzees of the intermediate size problem. *J. Exper. Psychol.*, 1942, 31, 257-271.
15. YOUTZ, R. E. P. The weakening of one Thorndikian response following the extinction of another. *J. Exper. Psychol.*, 1939, 24, 294-304.

CHAPTER XVI

Inhibition and Effective Reaction Potential

In the last chapter we considered experimental extinction as the mechanism which protects organisms from the evil effects of the unadaptive habits inevitably set up by the law of reinforcement. In the present chapter we shall again take experimental extinction as our point of departure; here, however, it will be regarded as a secondary phenomenon which arises under certain special conditions from the logically more primitive principle of *reactive inhibition*. Our expository procedure will be to state in a semi-formal manner certain principles, more or less physiological or submolar in nature, according to which reactive inhibition is believed to originate, operate, and disintegrate, and to accompany them with illustrative evidence. These submolar principles, it is to be noted, are not properly a part of the present system, which is molar, but are intended as a kind of background. Following this there will be presented a series of corollaries flowing from these and other principles of the system.¹ In this way an attempt will be made to show how the principles explain, and therefore integrate, an appreciable variety of relevant empirical phenomena. Finally two primary molar principles will emerge and will be formally stated as such at the end of the chapter.

QUANTITATIVE CONCEPTS AND PRELIMINARY STATEMENT OF PRIMARY MOLAR AND SUBMOLAR PRINCIPLES RELATED TO INHIBITORY POTENTIAL (I_R)

Although the physiology of response inhibition is far from clearly known, a great deal of knowledge of a submolar nature has been discovered during the last quarter century. Our account of this subject will therefore proceed at first with submolar principles as

¹For expository purposes the preliminary propositions and the corollaries which flow from them are more or less alternated in the following pages. It will be noted that the preliminary propositions are indicated by capital letters, whereas the corollaries are indicated, as in other chapters, by Roman numerals.

a background, the Mowrer-Miller hypothesis¹ being taken as a point of departure. While this hypothesis has a number of components which appear in various parts of the present chapter, the main or critical proposition may be stated as our first preliminary or submolar principle.

A. Whenever any reaction is evoked in an organism there is left a condition or state which acts as a primary negative motivation in that it has an innate capacity to produce a cessation of the activity which produced the state.

We shall call this state or condition *reactive inhibition*. From a quantitative point of view, reactive inhibition will be represented by the symbol I_R . Just as sE_R symbolizes a certain quantity of reaction evocation potential, so I_R symbolizes a certain potentiality of inhibition, i.e., a certain quantity of *inhibitory potential*. The reaction decrement which we have attributed to reactive inhibition obviously bears a striking resemblance to the decrements which are ordinarily attributed to "fatigue." It is important to note that "fatigue" is to be understood in the present context as denoting a decrement in action evocation potentiality, rather than an exhaustion of the energy available to the reacting organ (17).

From the foregoing it is evident that inhibitory potential (I_R) is an unobservable and so has the status of a logical construct with all the advantages and disadvantages characteristic of such scientific concepts. In this connection it will be recalled (p. 21 ff.) that the prime prerequisite for the proper use of unobservables in scientific theory is that they be anchored in a quantitatively unambiguous manner (a) to observable antecedent conditions or events, and (b) to observable consequent conditions or events.

Proceeding at once to the satisfaction of the first of these requirements² in so far as the present state of our ignorance per-

¹ A brief statement of Dr. Mowrer's version of the Mowrer-Miller hypothesis is contained in an article by himself and Miss Jones (15); Dr. Miller's version is presented in his recently published book (14, p. 40 ff.). We are much indebted to Dr. Mowrer not only for material appearing in the present section, but for ideas scattered throughout the entire chapter; however, for the particular formulation of the hypothesis here presented, in so far as it differs from the views of Dr. Mowrer and Dr. Miller, as well as for the deduction of the most of the corollaries derived in one way or another from it, the present author takes entire responsibility.

² The second requirement not only of the present unobservable but of a number of others employed in earlier chapters will be taken up in Chapter XVIII.

mits, we arrive at our second preliminary or submolar proposition:

B. The net amount of functioning inhibitory potential resulting from a sequence of reaction evocations is a positively accelerated function of the amount of work (W) involved in the performance of the response in question.

Stated still more specifically, it may be said that the weight of the evidence at present available indicates that

$$I_R = \frac{cn}{B - W},$$

where n is the number of reaction evocations involved, c and B are empirical constants, and

$$W = F'L,$$

in which F' represents force and L represents distance or length of the movement, as in ordinary mechanics.

It is evident that the mean net increment of inhibition per reinforcement must be the net inhibition divided by the number of reaction evocations. From this consideration and the basic equation for I , it follows that for a given organism the mean net increment of inhibition must have a constant value for a given value of W , i.e., it must be

$$B - W$$

It is to be noted in this connection, however, that the inhibitory potential resulting from a series of motor responses is not a simple matter of mechanics, that it does not depend merely upon the force (F') and distance (L) involved in the movement. This is precluded by the constant, c , in the relationship. For example, it is presumable that for a given amount of energy consumption such as is required for the repeated lifting of a heavy weight, the value of c would be larger for the weak muscular system involved in the flexing of the little finger than for the relatively strong muscular system which flexes the arm at the elbow.

The relationship of energy expenditure or work (W) to the accumulated inhibition I_R arising from a sequence of unreinforced reaction evocations, such as occur in experimental extinction, is convincingly demonstrated in an investigation reported by Mowrer and Jones (15). Three comparable groups of albino rats were trained on a Skinner type of apparatus to press a bar for food

pellets. The bar was so constructed that different weights could be attached to it requiring the animal to make any desired minimal pressure before the food would be delivered. The animals of all three groups were given equal preliminary reinforcement with bar weights of 5 grams, 42.5 grams, and 80 grams. Following this the three groups were extinguished during three periods of 20 minutes each, with 24 hours intervening between each extinction period.

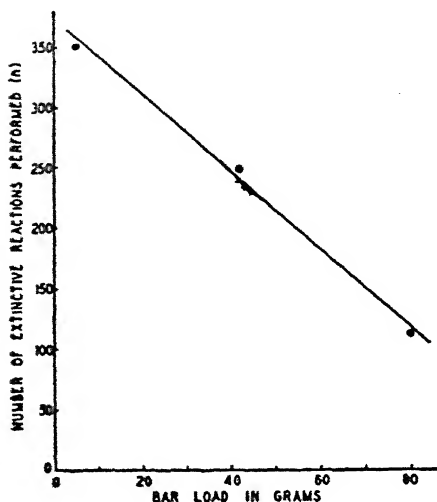


Fig. 63. Graph showing the relationship of the number of unreinforced reactions performed by albino rats in one hour to the amount of work involved in each reaction. (Plotted from data published by Mowrer and Jones, 15.)

One group had the bar weighted throughout the process exclusively with 5 grams; the second group had the bar weighted with 42.5 grams; and the third group had the bar weighted with 80 grams. The mean number of unreinforced reactions made by each of the respective groups of animals is shown in Figure 63. There it may be seen that the number of extinctive reactions performed under constant conditions of habit strength and motivation is approximately an inverse linear function of the work involved in the act. Evidence reported by Crutchfield (2), from a rather different ex-

perimental situation, also suggests an inverse linear relationship. This relationship may be expressed rather precisely by a transposition of the expression for I given above, i.e.,

$$n = \frac{I_R(B - W)}{c}$$

Since we are committed to a centigrade scale in the present system, it follows that the maximum of inhibitory potential must arbitrarily have a value of 100. Accordingly we take the unit of inhibitory potential as that amount of I_R which will just neutralize one unit of reaction potential. This unit will be called the *pav*, a syllable from the name Pavlov. It is suggested that the term *pav*

be pronounced to rhyme with *have*. The pav is accordingly defined in terms of the wat, thus:

$$1 \text{ wat} + 1 \text{ pav} = 0.^1$$

Having formally specified the value of I_R , we must now inquire what changes take place in it with the passage of time. It will be recalled that inhibitory potential is assumed on the submolar level to have its physical basis in a negative motivational condition or state. This quite probably depends on a substance resident in the effector organs involved in the response. Now, it is to be expected that such a substance will gradually be removed by the blood stream passing through these organs. Moreover, the amount of this removal per unit time following the cessation of the action should be proportional to the amount of inhibitory substance present at any given time. This, of course, is equivalent to saying that the dissipation of I_R will take place according to a simple decay or negative growth function (p. 199 ff.) of time. We thus arrive at our third preliminary proposition:

C. Each amount of inhibitory potential (I_R) diminishes progressively with the passage of time according to a simple decay or negative growth function.

THE CONDITIONING OF INHIBITORY POTENTIAL, ITS STIMULUS GENERALIZATION, AND THE CONCEPT OF EFFECTIVE REACTION POTENTIAL

At this point in our analysis we need to emphasize a somewhat different aspect of the above principles from that employed in the derivation of the preceding two propositions. The new emphasis will be on that portion of the Mowrer-Miller hypothesis (preliminary Proposition A) which states that the after-effect of reaction evocation is a primary negative motivational state or condition. This means that the after-effects of response evocation in the aggregate constitute a negative drive strongly akin to tissue injury or "pain." If this is the case, we should expect that the cessation of the "nocuous" stimulation in question or the reduction in the

¹It must be observed that the formal precision of the definition of the unit of inhibition is superficially deceptive in that it is programmatic rather than an accomplished fact. This statement holds for the units of habit strength and all similar units employed in the present work. It is believed that the use of such units even in a merely formal and programmatic sense adds to the clarity of the exposition and will contribute ultimately to an empirically workable operational definition.

inhibitory substance, or both, would constitute a reinforcing state of affairs. The response process which would be most closely associated with such a reinforcing state of affairs would obviously be the cessation of the activity itself. In accordance with the "law of reinforcement" (p. 80 ff.) this cessation of activity would be conditioned to any afferent stimulus impulses, or stimulus traces, which chanced to be present at the time the need decrement occurred. Consequently there would arise the somewhat paradoxical phenomenon of a negative habit, i.e., a habit of *not* doing something (14, p. 40 ff.). Thus we arrive at our first corollary:

I. *Stimuli closely associated with the acquisition and accumulation of inhibitory potential (I_R) become conditioned to it in such a way that when such stimuli later precede or occur simultaneously with stimulus situations otherwise evoking positive reactions, these latter excitatory tendencies will be weakened.*

Fortunately the existence of such habits is well authenticated, having long ago been demonstrated experimentally by Pavlov in what he called *conditioned inhibition*. Pavlov reports (16, p. 77 ff.) that a tactile stimulus was conditioned in a dog to a defensive salivary secretion produced by an injection of weak acid into the animal's mouth. In addition, an alimentary conditioned reflex was set up to the ticking of a metronome by having the latter followed by feeding. Then the metronome and a neutral stimulus in the form of a whistle were repeatedly presented together without reinforcement, which produced experimental extinction of the conditioned alimentary reaction. Now, according to the present hypothesis this extinction process should become conditioned to any neutral stimuli associated with it, notably the whistle; the remainder of the experiment proved this to be the case. After presenting the tactile stimulus a couple of times to demonstrate the strength of its power to evoke the salivary reaction, the experimenter presented it for the first time in conjunction with the whistle. The quantitative results are shown in Table 8. There it may be seen that at 3:16 the tactile stimulus evoked a secretion of 8 drops in one minute, whereas at 3:25, when the tactile stimulation was combined with the whistle, the secretion was less than one drop. At 3:30 the tactile stimulus, now applied alone, evoked 11 drops, which still further emphasizes the inhibitory effects of the whistle in the stimulus combination presented at 3:25. It had, of course, been demonstrated that previous to association with the extinction of the metronome-salivary conditioned reaction the whistle did not

TABLE 8

THIS TABLE PRESENTS EXPERIMENTAL RESULTS ILLUSTRATING THE ACTION OF CONDITIONED INHIBITION (EVOKED BY A WHISTLE) IN LARGELY SUPPRESSING THE EVOCATION OF A HETEROGENEOUS CONDITIONED REACTION. (From Pavlov, 16, p. 77.)

Time	Stimulus Applied During 1 Minute	Salivary Secretion in Drops During 1 Minute
3:08 P.M.	tactile.....	3
3:16 P.M.	tactile.....	8
3:25 P.M.	tactile + whistle.....	less than 1 drop
3:30 P.M.	tactile.....	11

produce external inhibition (16, p. 77) of the tactile-evoked salivary reaction.

In continuing the discussion of conditioned inhibition it must be pointed out that Corollary I has introduced a new dimension into the concept of inhibitory dynamics; this is to the effect that the influence of inhibition on behavior evocation may be controlled by a stimulus. Such a possibility requires the employment of a new symbol which will explicitly express this fact. We shall do this simply by adding to the symbol for ordinary inhibitory potential, I_R , the letter S as an extra subscript, thus: ${}_SI_R$.

But the moment the action of inhibitory potential (${}_SI_R$) is cued to a stimulus, the suggestion arises from the analogy to ${}_SH_R$, and so to ${}_SE_R$, that inhibitory potential will also manifest stimulus generalization; this brings us to our second corollary:

II. *Conditioned inhibitory potential (${}_SI_R$) will manifest stimulus generalization in a manner exactly analogous to that of reaction potentiality (${}_SE_R$), as given in Postulate 5.*

The appearance of two forms of inhibition on the theoretical scene instantly raises the question as to how they combine, which, in turn, requires the introduction of the concept of *total inhibitory potential*; this will be represented by the symbol \dot{I}_R . In this way we arrive at the statement of our fourth preliminary proposition:

D. Simple reactive inhibition (I_R) and conditioned inhibition (${}_SI_R$) summate functionally to produce \dot{I}_R as would corresponding amounts of habit strength (p. 223).

With the concept of total inhibitory potential available, it becomes necessary to introduce explicitly the concept of *effective reaction potential*; this is represented by the symbol ${}_SE_R$. The intro-

duction of this concept brings us to the statement of our fifth preliminary proposition:

E. The effective reaction potential (${}_s\bar{E}_R$), i.e., that reaction potential which is actually available for the evocation of action (R), is the reaction potential (${}_sE_R$) less the total inhibitory potential (\dot{I}_R).

Finally, with Proposition *E* available it becomes possible to derive the empirically known law of *spontaneous recovery*. This concept was employed by Pavlov (16, p. 58) to designate the well-known fact (see p. 271) that conditioned reactions which had suffered experimental extinction tended in the course of time spontaneously to recover a considerable proportion of their original effective reaction potentiality. By Proposition *C*, the inhibitory potential (I_R) operating against any given response (R) disintegrates according to a simple negative growth function. But since (Proposition *D*) \dot{I}_R is a summation of I_R and ${}_sI_R$, and since (Proposition *E*),

$${}_s\bar{E}_R = {}_sE_R - \dot{I}_R,$$

it follows that ${}_s\bar{E}_R$ will increase as \dot{I}_R decreases, which brings us to our third corollary:

III. *Other things equal, an effective reaction potential (${}_s\bar{E}_R$) which has been reduced by the accumulation of inhibitory potential (I_R) will recover spontaneously through the mere passage of time, the course of the recovery being a simple positive growth function of the time elapsing since the termination of the final response of the series which produced the inhibition in question.*

Ample verification of Corollary III in the case of extinctive inhibition was seen above (p. 270 ff. and Figure 62).

THE INCOMPLETENESS OF THE STIMULUS GENERALIZATION AND OF THE SPONTANEOUS RECOVERY OF EXTINCTIVE INHIBITION

Now let it be assumed explicitly, as was done tacitly in the derivation of Corollary I, that whereas ${}_sI_R$ involves the whole neural receptor-effector mechanism of habit, I_R involves only (or mainly) the effector portion of this mechanism. Accordingly it is to be expected that I_R would not manifest the generalization gradient characteristic of habit, but would display its presence by a constant amount of diminished effective reaction potential of the effector regardless of the stimulus evoking the reaction. This con-

stant or non-diminishing amount of reactive inhibition at all points on the stimulus generalization gradient of inhibitory potential brings us to our fourth corollary:

IV. *When a habit has been set up by well-distributed reinforcements and extinguished by massed evocations, the asymptote or limit of rise of effective reaction potential ($s\bar{E}_R$), due to the stimulus generalization of the conditioned inhibition, will always be less than the strength of the effective reaction potential just previous to the extinction.*

A second implication arising from the differential characteristics of I_R and sI_R hinges on the empirically established principle that in animal experimentation habits are relatively immune to forgetting, whether set up by the conditioned reaction technique or by selective learning (?). This means that (Proposition C) I_R will manifest the phenomenon of spontaneous dissipation as a function of time, whereas sI_R , being a habit, will not to any great extent. These considerations, coupled with Corollary III and the equation,

$$\dot{I}_R = I_R + sI_R,^1$$

bring us to the conclusion that spontaneous recovery of $s\bar{E}_R$ will occur only in so far as the extinctive inhibition is comprised of I_R . Thus we arrive at our fifth corollary:

V. *In case a reaction potential (sE_R) has been set up by distributed reinforcements and extinguished by massed evocations, spontaneous recovery of $s\bar{E}_R$ will be incomplete.* This we have seen to be true in the case of Ellson's investigation (lower curve, Figure 62).

But since stimulus generalization is based on sI_R or the habit aspect of reactive inhibition, it is not to be expected that the purely stimulus generalization of extinctive inhibition would show spontaneous recovery; this brings us to our sixth corollary:

VI. *Where effective reaction potential ($s\bar{E}_R$) falls below simple reaction potential (sE_R) by reason of purely stimulus generalization, i.e., from the action of conditioned inhibitory potential (sI_R), the effective reaction potential ($s\bar{E}_R$) will display no spontaneous recovery whatever.* No evidence bearing directly on Corollary VI has been found. An experimental test of its soundness would

¹ The sign + indicates physiological summation according to equations 32 and 43.

accordingly be of special value in determining the validity of the numerous assumptions ultimately involved in its derivation.

If, however, the difference between sE_R and $s\bar{E}_R$ is due to purely reactive inhibition, i.e., to the action of I_R , then complete spontaneous recovery should take place; which brings us to our seventh corollary:

VII. *Where effective reaction potential ($s\bar{E}_R$) falls below simple reaction potential (sE_R) by reason of the purely response generalization of extinctive inhibition, spontaneous recovery will occur and it will be complete.*

The spontaneous recovery observed in one portion of Ellson's experiment (3), that represented by the upper curve of Figure 62 (p. 271), presumably took place under conditions which approached those of response generalization. Accordingly it may, or may not, be significant for the validity of Corollary VII that spontaneous recovery was approximately perfect.

THE SUCCESSIVE EXTINCTION OF THE SAME REACTION POTENTIAL

From Corollary I it follows that I_R and sI_R are generated concurrently. In cases where experimental extinction is complete, i.e., where (Propositions *D* and *E*),

$$s\bar{E}_R = sE_R - (I_R + sI_R) = 0,$$

it follows that

$$sE_R = I_R + sI_R$$

This raises many intriguing questions as to the relative amounts of the two supposed forms of inhibitory potential that are generated under various conditions. As yet little experimental evidence concerning this matter exists, although Ellson's results suggest that during the initial extinction the two forms of inhibition are generated in roughly equal amounts.

From the preceding considerations it may be concluded that if an organism is subjected to massed extinctive evocations every five or six hours, say, there will be an appreciable amount of both I_R and sI_R generated in the first extinction. Six hours later the I_R will largely have been dissipated, but, since habits do not disintegrate with the mere passage of time, the sI_R will remain, so there will be an appreciably diminished amount of reaction potentiality

available for a second extinction; this will again generate both I_R and sI_R . But since there will be less sE_R to be extinguished, less of both I_R and sI_R will be generated than on the first occasion, so that fewer extinction evocations and less time will be required. Since there will be less and less I_R generated at each new extinction, there will be less and less spontaneous recovery after each recovery period. Thus we arrive at our eighth corollary:

VIII. *In case a reaction tendency (sE_R) is subjected to the same criterion of experimental extinction by massed evocations at uniform intervals, the amount of spontaneous recovery manifest at each successive extinction will progressively diminish until ultimately there may be no spontaneous recovery whatever, the number of unreinforced evocations required to produce a given degree of experimental extinction on the successive occasions being approximately a negative growth function of the ordinal number of the extinction in question.*

The evidence bearing on the soundness of Corollary VIII appears to be internally inconsistent. The necessities of adaptive dynamics demand that an organism shall not continue forever to waste its energy performing acts which yield no need reduction; this is in agreement with the general observation that organisms do in fact ultimately give up completely the performance of such acts. On the other hand, there appear to be situations, notably certain ones involving secondary reinforcement (p. 84 ff.), in which a considerable amount of spontaneous recovery continues to occur very many times. A striking case in point has been reported by Fitts (4). This investigator extinguished rats on a Skinner bar-pressing habit repeatedly at intervals of a week or longer. He found that the first three extinctions followed approximately the course deduced above, but the fourth extinction showed a statistically reliable increase in recovery which persisted, though with a gradual diminution, for five further extinctions. It seems likely that this reversal in Fitts' curve is due to some complex secondary reinforcement mechanism involving the stimuli arising from fractional antedating goal reactions, though the mechanism itself has not yet been worked out in detail.

THE PHENOMENON OF DISINHIBITION

If it is true, as implied by Corollary I, that conditioned inhibition (sI_R) is a negative habit, it is to be expected that the occur-

rence of an *extra stimulus* (an unaccustomed stimulus element) in the stimulus compound (S) would, through afferent neural interaction (p. 42 ff.), produce a diminution in the sI_R . This kind of causal mechanism is, of course, exactly that which presumably gives rise to what Pavlov calls external inhibition (see p. 217 ff.). When applied to inhibition itself, Pavlov calls the action of the extra stimulus *disinhibition* (16, p. 61 ff.). This brings us to our ninth corollary:

IX. *Whenever a stimulus element not customarily present in a compound stimulus (S) conditioned to an inhibitory tendency (sI_R) occurs in such a compound, the amount of inhibitory potential evokable by the new combination will be less than that normally evoked by the stimulus compound originally conditioned to the inhibition.*

Since any change in the inhibitory potential can become manifest only indirectly through positive action of some sort, it follows from the relationship,

$$s\bar{E}_R = sE_R - (I_R + sI_R),$$

that if the extra stimulus produced the same amount of reduction in sE_R as in sI_R , the two effects would exactly offset each other; i.e., no change whatever could occur in $s\bar{E}_R$, and therefore no change in observable behavior could result. Nevertheless, disinhibition is a well-authenticated empirical phenomenon. This seems to require the assumption of a special susceptibility of conditioned inhibition to being upset by extra stimuli. In this connection Pavlov remarks that

. . . the inhibitory process is more labile and more easily affected than the excitatory process, being influenced by stimuli of much weaker physiological strength. (16, p. 99.)

He cites experimental evidence which purports to substantiate this view; i.e., which shows that a weak stimulus will weaken the sI_R only, whereas a stimulation which includes a strong "extra" stimulus element may evoke no reaction whatever since it would also abolish the sH_R , and so the sE_R , by "external" inhibition.

If Pavlov's assumption of the differential action of weak extra stimuli on sI_R and sE_R is accepted, an important implication follows at once from the relationship,

$$s\bar{E}_R = sE_R - (I_R + sI_R).$$

Thus we arrive at our tenth corollary:

X. If a reaction potential (sE_R) has been partially or wholly extinguished, the inclusion of a mild extra stimulus in the conditioned stimulus compound (S) will result in the strengthening of the effective reaction potentiality ($s\bar{E}_R$).

But since sI_R constitutes only a portion of the total inhibition (I_R) which weakens sE_R , it follows that disinhibition can only partially restore $s\bar{E}_R$ to the original value of sE_R ; this leads to our eleventh corollary:

XI. When an excitatory habit has been set up by means of well-distributed reinforcements and has then been extinguished, the most effective disinhibitory stimulus possible will never (except through "oscillation"—see p. 304 ff.) enable S to evoke an R with as great vigor, certainty, or speed as before the extinction occurred.

INHIBITION OF REINFORCEMENT

It is evident from the above version of the Mowrer-Miller hypothesis (Proposition A) that reactive inhibition must be generated whenever reactions are evoked, whether reinforcement occurs or not. If reinforcement does not occur, the inhibitory potential generated by the response may be called *extinctive inhibition*; the inhibition generated if the response is followed by reinforcement has been called by Hovland, *inhibition of reinforcement* (8), from the circumstances of its origin, even though the inhibition is presumably not dependent upon the reinforcement process. If reinforcement occurs, the consequent increase in habit strength (sH_R) will so increase the reaction potential (sE_R) that when the inhibitory potential (I_R) arising from the successive reactions is deducted, the effective reaction potential ($s\bar{E}_R$) will usually be superthreshold in amount, i.e., more than enough, given normal stimulation and motivation, to evoke the reaction.

Now, inhibitory potential can be observed only indirectly through the failure to occur of some positive reactions which the antecedent conditions would otherwise produce. Because of this fact and of the usual over-riding effects of reinforcement, it happens that inhibition of reinforcement usually does not manifest itself in any very dramatic manner such, for example, as in the total cessation of reaction evocation so characteristic of experimental extinction. Probably it is because of these circumstances that relatively few investigators have noticed it. As might be ex-

pected, it was Pavlov who seems first to have observed and described this phenomenon. He remarks:

The development of inhibition in the case of conditioned reflexes which remain without reinforcement must be considered only as a special instance of a more general case, since a state of inhibition can develop also when the conditioned reflexes are reinforced. The cortical cells under the influence of the conditioned stimulus always tend to pass, though sometimes very slowly, into a state of inhibition. . . . This inhibition is of the same character as the internal inhibition which has been described in previous lectures, and it exhibits the same properties of irradiating to other cortical elements which are not primarily involved. (16, pp. 234-248)

In the above context Pavlov describes an illustrative experiment in which a conditioned reflex in the course of a number of rein-

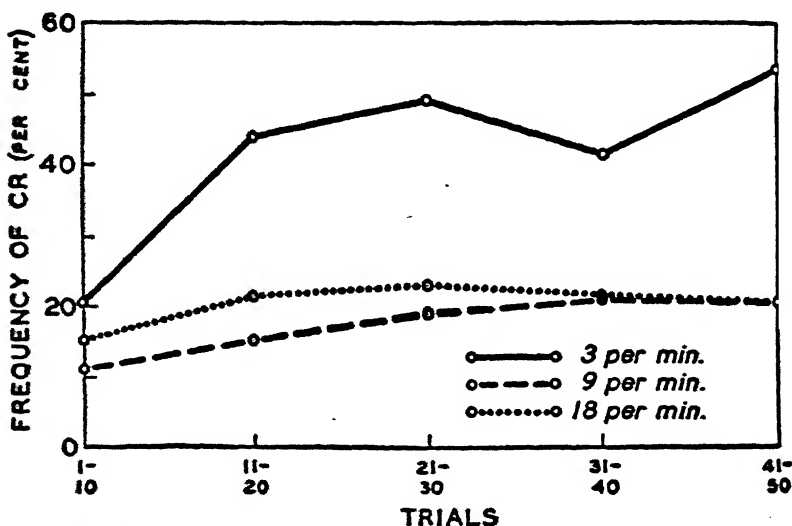


FIG. 64. Graphs showing the course of the acquisition of the conditioned lid reflex as a function of the time interval separating the reinforcements. (From Calvin, 1, as presented by Hilgard and Marquis, 7, p. 149.)

forcements given rather close together actually diminished to a zero strength (see Figure 64).

Thus we arrive at our twelfth and thirteenth corollaries:

XII. *Whenever conditioned reactions are evoked, whether reinforced or not, reactive inhibition (I_R) is generated.*

XIII. *In the case of closely massed reinforcements, the curve of acquisition of effective excitatory potential ($s\bar{E}_R$), particularly*

in its later stages, will be distorted by inhibition of reinforcement below the learning curve of sE_R , in extreme cases showing an actual fall with continued practice.

Calvin reports such a curve (Figure 64), in which the reinforcements occurred at the rate of eighteen times per minute; this curve not only ceases to rise after about 25 reinforcements, but actually shows a slight fall.

DISINHIBITION AND THE INITIAL RISE IN THE CURVE OF EXPERIMENTAL EXTINCTION

In 1930 Switzer (18) reported a novel form of experimental extinction curve, based on the extinction of the conditioned lid reaction. Instead of falling abruptly from the initial unreinforced reaction as in Figure 57, the curve showed at first a sharp rise in amplitude of reaction (Figure 65). After one or two more unreinforced reactions this initial rise was followed by the fall usually encountered in the extinction process. Hudgins (9; 10, p. 439) and others have fully verified Switzer's original discovery. Hovland (8) has reported the outcome of an ingenious experiment which he interprets as explaining the phenomenon found by Switzer. The habit involved was a galvanic skin

12
10
8

1 2 3 4 5 6 7
ORDINAL NO. OF EXTINCTION TRIALS

FIG. 65. Graph showing the Switzer phenomenon. Note the initial rise in the composite curve of experimental extinction of a conditioned eyelid reaction. (Plotted from pooled values published by Switzer, 18, p. 86.)

reaction produced by an electric shock which had been conditioned to an auditory stimulus. The results of the experiment are shown concisely by the four graphs appearing in Figure 66, graphs B and C being of special significance. Graph B, which is a curve of experimental extinction following massed reinforcements, shows what purports to be the Switzer effect; graph C is a curve of experimental extinction following a form of distributed reinforcements and shows a close approximation to the conventional curve of extinction. Hov-

land interprets these results as indicating (a) that massed reinforcements in considerable numbers leave at their conclusion a relatively large amount of "inhibition of reinforcement"; (b) that the transition from reinforcement to non-reinforcement acts as a disinhibiting agent, producing disinhibition of the accumulated inhibi-

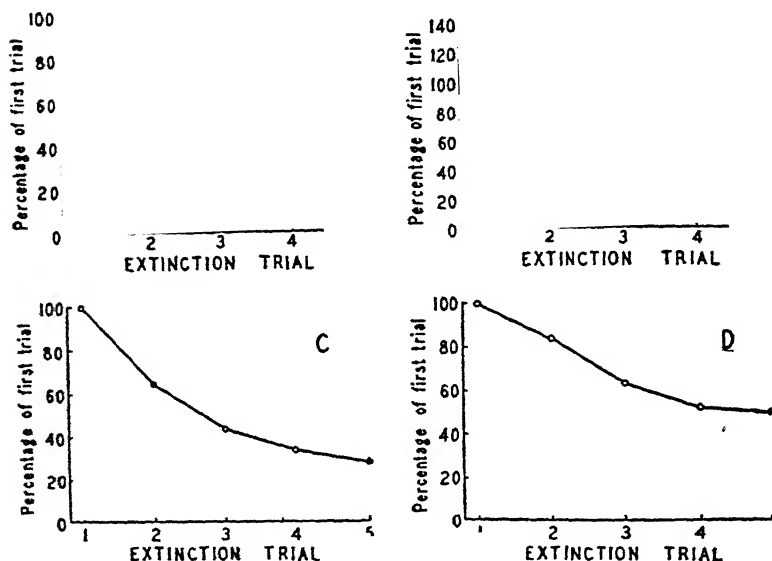


FIG. 66. Extinction curves following various conditions of reinforcement. Results plotted in terms of ratios (in per cent) of responses on successive extinction trials to response on first extinction trial. (A) 8 reinforcements. Extinction immediately. (B) 24 reinforcements. Extinction immediately. (C) 24 reinforcements, distributed into 3 groups of 8 each. Rest period of 30 minutes between groups. Extinction immediately after last group of reinforcements. (D) 24 reinforcements. Extinction 30 minutes after last reinforcement. (Reproduced from Hovland, 8, p. 431.)

tion of reinforcement. This accounts for the initial rise shown in graph B.

The set of assumptions outlined in the preceding pages of the present chapter imply the initial rise of the curve of experimental extinction in the following manner: (a) massed reinforcements generate a relatively large amount of I_R and consequently a strong negative drive; (b) each pause between reinforcements, even if brief, produces a slight reduction in the need (for inactivity, or rest); (c) this serves as a reinforcing state of affairs setting up

a certain amount of *conditioned* inhibition (sI_R); (d) the sudden transition from reinforcement to non-reinforcement, on the first non-reinforced stimulation, withdraws from the afferent impulses which customarily were present at previous reinforcements, the stimulus traces of the shock; (e) this change in the make-up of the afferent compound, through the principle of afferent interaction (p. 42 ff.), is sufficient to produce disinhibition of the conditioned inhibition of reinforcement; which (f) results in the initial rise in the curve of experimental extinction. Thus we arrive at our fourteenth corollary:

XIV. *When conditioned reactions are set up by means of massed reinforcements, conditioned inhibition is generated which, at the outset of extinction, is disinhibited through the change in the functioning afferent impulses, with the result that the curve of experimental extinction shows an initial rise.*

While Corollary XIV agrees in the main with Hovland's experimental findings, there are certain respects in which it does not. The greatest single inconsistency is shown in graph *D*, which represents the extinction of a conditioned reaction based on 24 massed reinforcements, 30 minutes after the conclusion of the reinforcement series. Since disinhibition is assumed to be based on sI_R , which is regarded as a habit, and since ordinary habits do not disintegrate appreciably in 30 minutes (7), there should have been about as much sI_R to be disinhibited in the case of graph *D* as in that of graph *B*, which clearly is not the case. The discrepancy is of considerable importance, since it points to a serious defect in the postulates which generate Corollary XIV. This matter clearly needs further intensive experimental investigation, particularly from the point of view of the assumed sI_R .

THE LAW OF LESS WORK

One of the traditional methods employed in the investigation of the gradient of reinforcement (p. 137 ff.) has been to give an organism the choice of two paths to the attainment of some sort of reinforcing agent, such as food, and study its behavior as reinforcements of the two behavior sequences accumulate. In one of the best and most recent investigations of this kind, Grice (6) concludes in effect that if the temporal factor (upon which the gradient of reinforcement is formulated) were completely equalized, there would still be a marked preference for the shorter path. In

of no practice there will exist a net increased effective reaction potentiality ($s\bar{E}_R$); this increase has received the not too appropriate designation of *reminiscence*. Thus we arrive at our eighteenth, nineteenth, twentieth, and twenty-first corollaries:

XVIII. *In case a simple conditioned reaction is set up to an appreciable strength by massed practice and the final reinforcement is followed by a no-practice period several times as long as the interval between reinforcements, after which the stimulus is again delivered, motivation remaining constant, the reaction-evocation potentiality of this stimulus will be greater than it was at the termination of the original reinforcement sequence.*

XIX. *In the case of simple conditioned reactions, reminiscence if plotted as a function of time will approximate a simple growth function.*

XX. *In the case of rote series learned by massed practice, reminiscence will rise at first with a negative acceleration, which will presently be replaced by a fall (11, pp. 261, 263, 277).*

XXI. *The closer the massing of the reinforcements in the original learning, the greater will be the extent of the reminiscence effect.*

The phenomenon of reminiscence in the conditioned reflex set up by means of massed practice was, as usual, first described by Pavlov (16, p. 249). A rather elaborate quantitative investigation of the phenomenon with human subjects, incidental to the study summarized in Figure 64, has been reported by Calvin (1). He found after a no-practice period of 24 hours a mean increase of from 5.75 reactions per ten trials to 7.10, a gain of 1.35 points, where the reinforcements were three per minute; where the reinforcements were eighteen per minute he found an increase of from 2.30 to 7.25 reactions, a gain of 4.95 points. This shows an appreciable advantage in reminiscence for the more closely massed reinforcements.

The phenomenon of reminiscence in rote learning has been known for many years. The last and most precise investigation of reminiscence in this form of learning was reported by Ward (21).

If we apply Corollary XVIII to any learning situation, it is evident that when reinforcements are separated by time intervals of moderate length, the greater these intervals, the greater will be the amount of spontaneous recovery during the intervals, i.e., the less the aggregate inhibitory potential at the end of learning, and therefore the greater will be the effective habit strength at the

conclusion of the last reinforcement. This is known in learning literature as "the economy of distributed repetitions." An excellent demonstration of this principle is furnished by the Calvin study (1) just referred to. Using human subjects, this investigator conditioned to a light stimulus the lid-closure reflex originally evoked by a shock below the eye. One group of 20 subjects received reinforcements at the rate of three per minute, another group at the rate of nine per minute, and a third group at the rate of eighteen per minute. The course of the learning in terms of the per cent of presentations of the conditioned stimulus evoking the conditioned reaction was as indicated in Figure 64. These curves show that the rate of learning where three reinforcements were given per minute was much faster than where reinforcements were given more closely massed. Many parallel experiments in various fields of learning, particularly in the rote learning of nonsense syllables (11, p. 127 ff.), have demonstrated the same type of economy.

From the preceding considerations we accordingly formulate our twenty-second corollary:

XXII. *Within limits, the greater the time interval separating the reinforcements of learning, the greater will be the effective excitatory potential ($s\bar{E}_R$) at the conclusion of the last reinforcement.*

SUMMARY

The Mowrer-Miller hypothesis states in effect that all responses leave behind in the physical structures involved in the evocation, a state or substance which acts directly to inhibit the evocation of the activity in question. The hypothetical inhibitory condition or substance is observable only through its effect upon positive reaction potentials. This negative action is here called reactive inhibition. An increment of reactive inhibition (ΔI_R) is assumed to be generated by every repetition of the response (R), whether reinforced or not, and these increments are assumed to accumulate except as they spontaneously disintegrate with the passage of time. The magnitude of the individual increments, and therefore of the rate of accumulation, appears clearly to be in part a positively accelerated increasing function of the amount of energy consumed by the response.

Because of the motivational characteristics of reactive inhibition, or inhibitory potential, it is opposed to reaction potential (sE_R) rather than to habit (sH_R), as is sometimes supposed. Thus

effective reaction potential (${}_s\bar{E}_R$), the potential actually available for the evocation of action, is the reaction potential less the inhibitory potential.

Since under ordinary learning conditions response and reinforcement occur in parallel, the strengthening of the habit due to reinforcement usually is great enough to over-ride the accumulating inhibition. As a consequence, inhibition of reinforcement is only detected by special means. In case little or no reinforcement follows the reaction evocations, extinctive inhibition soon neutralizes the reaction potential, the stimulus gradually ceases to evoke the response, and there ensues the state known as experimental extinction which thus appears as a secondary or derived phenomenon.

The Mowrer-Miller hypothesis regards reactive inhibition as essentially a need to cease action, i.e., a need for rest; it follows that anything which reduces this need should serve as a reinforcing state of affairs. Since the cessation of action reduces the afferent proprioceptive impulses generated by it in the presence of the inhibiting condition, particularly when many responses have generated a considerable amount of inhibition, it comes about that the *cessation* of action, rather than action, becomes conditioned to whatever stimuli may be present. In this way we find a plausible explanation of conditioned inhibition (${}_sI_R$) and of the stimulus generalization of extinction effects. There are a number of indications that phenomena analogous to conditioned inhibition and stimulus generalization of inhibition occur under conditions of ordinary learning reinforcements, though not all the empirical evidence harmonizes with this *a priori* expectation. For this reason the theory of the origin of ${}_sI_R$ must be regarded with somewhat more than the usual amount of distrust.

Because conditioned inhibition (${}_sI_R$) is generated as a secondary effect from the accumulation of reactive inhibition (I_R), it follows that at least in extinction situations both I_R and ${}_sI_R$ will result. Assuming that the two summate physiologically, it follows that at complete experimental extinction the excitatory potential (${}_sE_E$) will be opposed or neutralized in part by I_R and in part by ${}_sI_R$. Now, I_R dissipates spontaneously through the passage of time, but ${}_sI_R$, being a true habit, presumably does not, at least to any great extent. The dissipation of I_R will produce spontaneous recovery of direct extinction effects, but this will naturally result in only partial recovery. On the other hand the second inhibitory component in

extinction (sI_R) should be subject to external inhibition. Since sI_R is responsible for only a portion of the depression of $s\bar{E}_R$ below sE_R , disinhibition, which presumably operates only on sI_R , also should never produce complete recovery. The slight initial rise in response vigor when extinction follows massed reinforcements is plausibly interpreted as the external inhibition of the conditioned inhibition (sI_R) presumably set up during the reinforcement process. The facts, however, are not wholly in harmony with this interpretation.

In case a reaction tendency (sE_R) is extinguished a good many times, each extinction being performed by massed practice on separate occasions, the gradual accumulation of the relatively permanent conditioned inhibition implies that the time required for the successive extinctions of the reaction tendency should grow less and less, the minimum approaching zero as a limit.

The magnitude of I_R , and also, presumably, of sI_R , generated by a given number of response evocations depends upon the amount of energy consumption or work (W) involved. This implies that of two or more alternative behavior sequences repeatedly executed by the organism in the attainment of an ordinary reinforcement, that sequence will finally come to be chosen which involves the less work or the less tissue injury. This is the important "law of less work," which, as pointed out by James (12), accounts for the prevalence of "laziness" in the behavior of organisms.

Because reactive inhibition (I_R) dissipates spontaneously through the passage of time, it follows that a part of the "inhibition of reinforcement" will dissipate during the pauses which occur throughout learning by distributed reinforcements or repetitions. The less the I_R , presumably, the less will be the sI_R , and so, certainly, the less the \dot{I}_R and consequently the greater will be the $s\bar{E}_R$ at the end of the learning process. Thus is explained the well-known empirical law of the economy of distributed repetitions in learning. If, on the other hand, a considerable number of reinforcements are massed and then a pause occurs, the same principle leads to the frequently observed empirical phenomenon of spontaneous recovery of effective reaction potential ($s\bar{E}_R$) known as "reminiscence."

In view of the considerations, molar and submolar, put forward in the preceding pages, we now formulate our eighth and ninth primary molar principles:

POSTULATE 8

Whenever a reaction (R) is evoked in an organism there is created as a result a primary negative drive (D); (a) this has an innate capacity (I_R) to inhibit the reaction potentiality (sE_R) to that response; (b) the amount of net inhibition (\bar{I}_R) generated by a sequence of reaction evocations is a simple linear increasing function of the number of evocations (n); and (c) it is a positively accelerated increasing function of the work (W) involved in the execution of the response; (d) reactive inhibition (I_R) spontaneously dissipates as a simple negative growth function of time (t''').

POSTULATE 9

Stimuli (S) closely associated with the cessation of a response (R) (a) become conditioned to the inhibition (I_R) associated with the evocation of that response, thereby generating conditioned inhibition; (b) conditioned inhibitions (sI_R) summate physiologically with reactive inhibition (I_R) against the reaction potentiality to a given response as positive habit tendencies summate with each other.

NOTES

Mathematical Statement of Postulate 8

$$(a) \quad s\bar{E}_R = sE_R - I_R \quad (36)$$

$$(b) \quad n = \frac{sI_R (B - W)}{c} \quad (37)$$

$$(c) \quad sI_R = \frac{cn}{B - W} \quad (38)$$

$$\text{where} \quad W = FL \quad (39)$$

$$(d) \quad e''' I_R = I_R e^{-ct'''} \quad (40)$$

Mathematical Statement of Postulate 9

$$(a) \quad \bar{I}_R = I_R + sI_R - \frac{I_R \times sI_R}{M_1} \quad (41)$$

$$(b) \quad s\bar{E}_R = sE_R - \bar{I}_R \quad (42)$$

The Mowrer-Jones Graph

is,

$$n = 373 - 3.2 \, gm,$$

where n is the number to produce extinction,
and gm is the pressure in grams required at each reaction.

It may be noted in this connection that the form of the above equation is not exactly that shown as (37) above. A transformation of the former equation to agree with (37) is,

$$x = \frac{116.6 - W}{.3125} \quad (43)$$

where 116.6 = B , and .3125

Equations Expressing the Laws of the Disintegration of Reactive Inhibition (I_R) and of the "Recovery" of Effective Reaction Potential ($s\bar{E}_R$)

The negative growth or decay principle according to which reactive inhibition is assumed to disintegrate (Proposition C) is:

$${}^{''''}I_R = I_R \times 10^{-t^{''''}}.$$

Since,

$$s\bar{E}_R = sE_R - (I_R + sI_R),$$

it follows that $s\bar{E}_R$ after the lapse of time, $t^{''''}$, will be,

$$\begin{aligned} {}^{''''}s\bar{E}_R &= sE_R - (I_R + sI_R) \\ &= sE_R - (I_R \cdot 10^{-t^{''''}} + sI_R), \end{aligned}$$

from which it follows that $s\bar{E}_R$ will with sufficient time "recover" substantially the amount to which it is depressed by I_R , but *not* the amount to which it is depressed by sI_R ; and the recovery will take place according to the exponential or positive growth function of time ($t^{''''}$).

Problems Connected with the Conditioning of Inhibitory Potential (sI_R)

The theory of the generation of sI_R from I_R presents a number of problems which probably cannot be cleared up without a considerable amount of co-ordinated research. One major problem may be stated as follows: If the cessation of contraction can serve as a reinforcing state of affairs, why does this not serve to set up habits involving muscular contraction, as well as inhibition? Such an implication seems to be perfectly legitimate, and it presents numerous intriguing possibilities. In this connection it is important to recall the nature of the gradient of reinforcement (p. 139 ff.), which is to the effect that the process most closely preceding the reinforcing state of affairs will be the one most strongly reinforced. On this principle, the cessation of the "nocuous" stimulation from a muscle will reinforce most strongly the cessation, relaxation, or inhibition of the act which produced the discharges and distinctly less the active contraction which necessarily has preceded the relaxation. Thus while some reinforcement of excitation leading to positive reaction potential (sE_R) would result from the cessation of "fatigue" stimulations arising from muscular action, a much greater, and therefore a dominating, amount of conditioned inhibitory potential (sI_R) would be generated.

A second and still more complex problem may be stated as follows: If the cessation, relaxation, or inhibition of action is susceptible to being conditioned to stimuli, as the phenomenon of conditioned inhibition in conditioned reflexes

strongly suggests, why does not an ordinary reinforcing agent, such as the reduction of the food need, reinforce inhibitory, quite as much as excitatory, tendencies?

While this problem requires a much more thorough examination than can be given here, a few suggestions may be made. In the first place, there is much evidence that conditioned inhibition is generated in extinction situations, and the initial rise of the extinction curve suggests that conditioned inhibition is also evolved in ordinary reinforcing situations. It is conceivable that some of this conditioned inhibition is set up by means of the mechanism just described. The critical question concerns the relative amounts of positive reinforcement (sH_R) and of negative reinforcement (sI_R) which will be generated according to the present set of hypotheses. If inhibitory tendencies were reinforced the same as excitatory tendencies, the two might simply neutralize each other, in which case no positive effective reaction potentials would develop, and effective learning could not occur. Such an outcome would be implied by the naïve supposition that reinforcement does not take place until after the cessation of the act reinforced. For example, in the Skinner procedure the animal does not eat until after the cessation of the muscular contraction which depresses the bar. Actually, while this is true of primary reinforcement, it is also true that a large portion of the reinforcement in such learning is secondary in nature, and this secondary reinforcement, e.g., the click of the magazine, occurs *during* the contraction and *preceding* the relaxation. In this connection it is well to recall evidence of the occurrence of reinforcement when the reinforcing state of affairs precedes the reaction reinforced. Both the work of Thorndike (19, p. 35) and that of Jenkins (13, pp. 58a and 72a), however, have shown that the forward wing of this double gradient is relatively much lower than the backward one. The difference in the strength of reinforcement in the two positions should give the conditioned excitatory tendency (sE_R) something like the advantage over the conditioned inhibitory tendency (sI_R) that experiment shows it to have in fact.

This question will evidently require much further investigation before a confident decision can safely be made regarding numerous aspects of reactive inhibition in adaptive behavior. Indeed, the present chapter may be considered to be largely an analytical exploration of a very rich field, preliminary to such a coordinated research program. It is also to be remembered that this uncertainty essentially concerns the submolar basis of Postulate 9; from a logical point of view all of this difficulty is eliminated when we take this proposition as a primary molar law, i.e., as a separate postulate. If, as seems likely, Postulate 9 is later rigorously derivable from other principles of the system, it will become a corollary, and the number of primary principles will thereby be reduced by one.

REFERENCES

1. CALVIN, J. S. Decremental factors in conditioned-response learning. Ph.D. thesis, Yale University, 1939.
2. CRUTCHFIELD, R. S. The determiners of energy expenditure in string-pulling by the rat. *J. Psychol.*, 1939, 7, 163-178.
3. ELLSON, D. G. Quantitative studies of the interaction of simple habits. I. Recovery from specific and generalized effects of extinction. *J. Exper. Psychol.*, 1938, 23, 339-358.
4. FITTS, P. M. Perseveration of non-rewarded behavior in relation to food-deprivation and work-requirement. *J. Genet. Psychol.*, 1940, 57, 165-191.

5. GENGHERELLI, J. A. The principle of maxima and minima in animal learning. *J. Comp. Psychol.*, 1930, 11, 193-236.
6. GRICE, G. R. An experimental study of the gradient of reinforcement in maze learning. *J. Exper. Psychol.*, 1942, 30, 475-489.
7. HILGARD, E. R., and MARQUIS, D. G. *Conditioning and learning*. New York: D. Appleton-Century Co., Inc., 1940.
8. HOVLAND, C. I. 'Inhibition of reinforcement' and phenomena of experimental extinction. *Proc. Natl. Acad. Sci.*, 1936, 22, No. 6, 430-433.
9. HUDGINS, C. V. Conditioning and the voluntary control of the pupillary light reflex. *J. Gen. Psychol.*, 1933, 8, 3-51.
10. HULL, C. L. Learning: II. The factor of the conditioned reflex. Chapter 9 in *A handbook of general experimental psychology*. Worcester, Mass.: Clark Univ. Press, 1934.
11. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
12. JAMES, W. T. Experimental observations indicating the significance of work on conditioned motor reactions. *J. Comp. Psychol.*, 1941, 32, 353-366.
13. JENKINS, W. O. Studies in the spread of effect. Ph.D. thesis, Yale University, 1942.
14. MILLER, N. E., and DOLLARD, J. *Social learning and imitation*. New Haven: Yale Univ. Press, 1941.
15. MOWLER, O. H., and JONES, H. M. Extinction and behavior variability as functions of effortfulness of task (in press).
16. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
17. ROBINSON, E. S. Work of the integrated organism. Chapter 12 in *A handbook of general experimental psychology*. Worcester, Mass.: Clark Univ. Press, 1934.
18. SWITZER, S. A. Backward conditioning of the lid reflex. *J. Exper. Psychol.*, 1930, 13, 76-97.
19. THORNDIKE, E. L. *The psychology of wants, interests, and attitudes*. New York: D. Appleton-Century Co., Inc., 1935.
20. TSAI, L. S. The laws of minimum effort and maximum satisfaction in animal behavior. *Monog. Natl. Res. Instit. Psychol.* (Peiping, China), 1932, No. 1. (See in *Psychol. Abstr.*, 1932, 6, 4329.)
21. WARD, L. B. Reminiscence and rote learning. *Psychol. Monog.*, 1937, 49, No. 4.
22. WATERS, R. H. The principle of least effort in learning. *J. Gen. Psychol.*, 1937, 16, 3-20.
23. WHEELER, R. H. *The science of psychology*. New York: Thomas Y. Crowell Co., 1929.

CHAPTER XVII

Behavioral Oscillation

It is an everyday observation that organisms vary in their performance even of well-established, habitual acts from occasion to occasion and from instant to instant on the same occasion. We are able to recall a name at one time but not at another; in shooting at a target we ring the bell at one shot, but not at the next; and so on.

When a six-place number is divided by a five-place number repeatedly by an automatic calculating machine, the exact identity of the quotient obtained on all the occasions is taken for granted; if the average person were to perform the same divisions, using pencil and paper, he would consider himself lucky if exactly the same result were obtained each time. While first-rate calculating machines sometimes get out of order and make errors, ordinary inorganic mechanisms under the same external conditions show, in general, much less variability in behavior than do organisms. Indeed, variability, inconsistency, and specific unpredictability of behavior have long been recognized as the chief molar distinctions between organisms and inorganic machines. Clearly a characteristic so fundamental as this must find an important place in any adequate theory of organismic behavior.

EXPERIMENTAL DEMONSTRATIONS OF BEHAVIORAL OSCILLATION

Even when the strength of a reaction potential has become stabilized at a value well above the reaction threshold, and the conditioned stimulus evokes its reaction with a considerable degree of consistency, both the amplitude and the latency of the reaction always oscillate from trial to trial. This is illustrated nicely by data from an unpublished study performed in the author's laboratory by Ruth Hays and Charles B. Woodbury. In this investigation a hungry albino rat was placed in a Skinner type of apparatus (Figure 61, p. 268), the single pressure bar of which was provided with a recording dynamometer. In the first part of the experiment the apparatus was so set that the animal was required to make a pressure of 21 grams before the food-pellet reward would

be delivered. Four days of training were then given, on each of which the animal received 100 reinforcements; on the day following, under exactly the same conditions, the animal made the distribu-

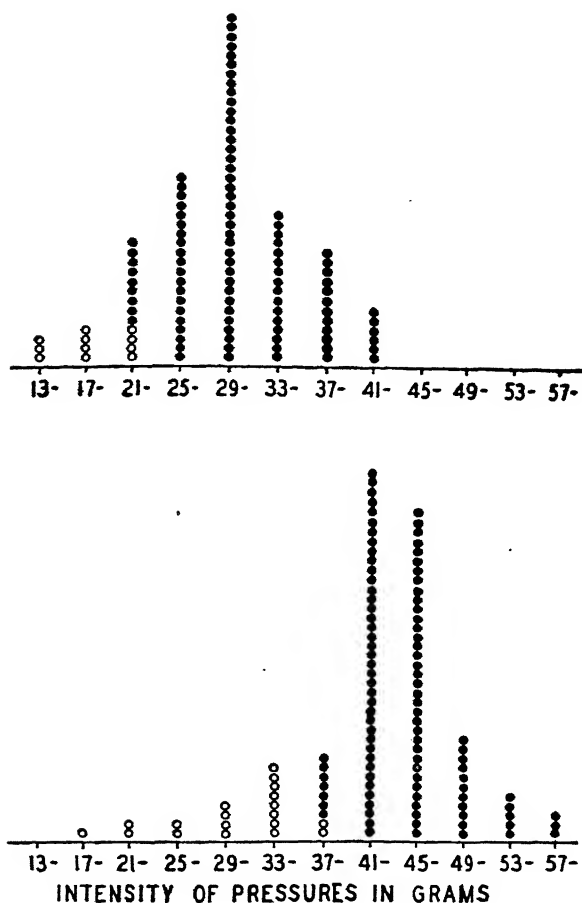


FIG. 67. Two reaction-intensity distributions of a rat in a bar-pressing experiment, designed to illustrate the oscillation of reaction potential (sE_R). See text for description of experimental procedure. (From an unpublished study performed in the author's laboratory by Ruth Hays and Charles B. Woodbury.)

tion of pressures shown in the upper portion of Figure 67. There the solid circles represent the pressures which were followed by food reinforcement, and the hollow circles represent the pressures which were too weak to deliver the food pellet. This distribution shows

the phenomenon of intensity variability of a single organism within a short period of time under practically identical external stimulating conditions; the maximum pressure was more than three times as great as the weakest, and about twice as great as the minimum required to yield food pellet delivery. The upper portion of Figure 67 also suggests that the distribution of oscillating reaction intensities conforms approximately to the normal "law" of chance.

Further illustration of the same general tendencies is presented by the lower distribution in Figure 67. This shows the variability of pressures by the same animal in the same apparatus after four more days of 100 reinforcements each with an apparatus adjustment which required the animal to make a pressure of 38 grams before the food pellet would be delivered. Here we find an even wider range of variability in pressure intensities than that shown in the upper distribution. Again there is a general tendency for the distribution to conform to the normal "law" of chance, though in this case the conformity is not so close as in the upper distribution. Several other animals trained like the one whose record has been given above, showed exactly similar tendencies in all respects. Moreover, Hill (3), in connection with an investigation directed at an entirely different objective, secured results on the conditioned eyelid reaction which incidentally showed that both amplitude and reaction latency under relatively constant conditions display an oscillation closely comparable to that revealed by the rat pressures of Figure 67.¹

Tables published by Thorndike (10) based on a line-drawing experiment where learning apparently was effectively precluded by human subjects whose eyes were closed, substantiate the results yielded by Hays and Woodbury's rats. Thorndike instructed his subjects to draw what seemed to them to be a two-inch line, for example. One subject drew 1,697 of these lines. This distribution shows great variability or oscillation, with the majority of the lengths falling in the central region much as shown in Figure 67. Thorndike's distribution revealed, however, a long, thin, asymmetrical tail on the side of the longer lines. Since the rat data mentioned above give no suggestion of this asymmetry, one may reasonably conjecture that it was produced by some factor in the experimental situation other than the primitive oscillation tendency. A further examination of Thorndike's tables reveals one

¹ Received in a private communication and reported here with Dr. Hill's

subject who drew a large number each of two-inch, four-inch, and six-inch lines. These comparable distributions indicate a progressive increase in the range of oscillation in the length of line drawn. Additional study shows that the range of oscillation in this series has an approximately linear relationship to the central tendency of the lines actually drawn. Here again there is essential agreement with the rat behavior shown in Figure 67.

A striking, though indirect, indication of the oscillation or

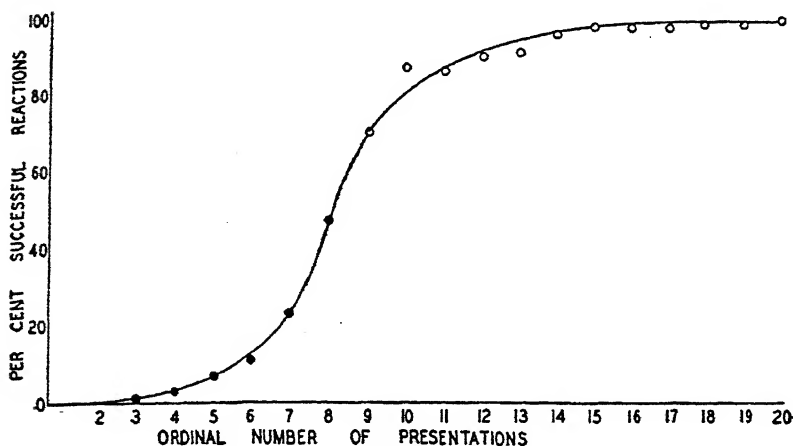


FIG. 68. Graphic representation of the empirical per cent of successful reactions at the presentation of the cue syllable in the case of the learning of 205 nonsense syllables in series. The complete learning of each of the syllables in question was preceded by six failures of reaction evocation (with reinforcement), i.e., six presentations of the cue syllable which were not followed by the correct response. (Reproduced from *Mathematico-Deductive Theory of Rote Learning*, 5, p. 162.)

variability of behavior potentiality is presented in the early stages of most simple conditioning situations where the experiment is so set up that conditioned and unconditioned reactions are clearly distinguishable. Under these circumstances it is common for the conditioned stimulus to evoke its reaction on one occasion, yet not on the next, in spite of the fact that because of intervening reinforcement the effective habit strength must have been stronger at the time the failure occurred than it was at the time of the preceding success (4).

Notwithstanding the seemingly fortuitous occurrence of reactions during the early stages in the acquisition of receptor-effector

connections, the probability of evocation actually increases continuously with the increase of the effective habit strength and, since drive is presumably constant, of effective reaction potential (${}_s\bar{E}_R$). This may be shown by pooling the evocation results mediated by a large number of ${}_sH_R$ connections which increase in strength at approximately the same rate. There is revealed by this procedure not only a progressive increase in the probability of reaction evocation but a characteristic sigmoid curve of increase. The outcome of such a procedure is shown in Figure 68. The normal chance distribution of Figure 67 in the learning situation just described would produce exactly the sigmoid learning curve seen in Figure 68.

The above results taken as a whole indicate that *the same external stimulating conditions, operating through an approximately identical habit structure (${}_s\bar{H}_R$) and a relatively constant drive (D), and so an approximately constant effective reaction potential (${}_s\bar{E}_R$), will evoke distinctly diverse reactions.*

ASYNCHRONISM OF BEHAVIORAL OSCILLATION

The phenomena represented in Figure 68 are presumably produced by the gradual rise of the effective strength of a habit above the reaction threshold. In such a situation, if the ${}_s\bar{E}_R$ chances to oscillate so as to be above the reaction threshold at the moment of stimulation, overt reaction will occur; if it chances to fall below the reaction threshold, the reaction will not occur. Reaction will follow *every* stimulation only on condition that the momentary effective reaction potential exceeds the reaction threshold by an amount greater than the range of oscillation below it.

There is a related case in which the problem is concerned not with the occurrence or non-occurrence of a reaction but with which one of two or more competing incompatible reactions¹ will be evoked by a given stimulus situation when the effective reaction potential of each exceeds the reaction threshold by an amount greater than the range of oscillation below it. Under such conditions each reaction tendency, if not interfered with by another incompatible one originating in the same stimulus situation, would mediate its reaction at every stimulation. Suppose, now, that to these conditions there be added a further one, namely, that the

¹ Incompatible reactions are those which cannot be executed at the same time.

habit strength of one of two competing reaction tendencies exceeds that of the other, but by an amount less than the range of oscillation below it. It follows that if the competing effective reaction tendencies both oscillate upward or downward at the same time, i.e., in synchronism, the one with the strongest habit strength will always dominate, its reaction occurring at every stimulation and the other reaction not occurring at all. If, however, the oscillations of the two reaction tendencies are asynchronous or at least are not perfectly correlated, then there may be expected an irregular alternation, the relative frequency of the occurrence of each reaction being an increasing function of the difference between the respective habit strengths (see Table 3 and Figure 36, pp. 147 and 150).

Experiment reveals the latter state of things rather than the former. It follows that in a trial-and-error learning situation of this kind, where the strength of one of several competing reaction tendencies is steadily increasing with respect to the others, dominance by this reaction tendency would be attained gradually rather than abruptly; this also is a fact. The outcome of such a process, where the ultimately dominant habit was at the outset relatively weak, is shown in Figure 24 (p. 108). It is abundantly clear that *the oscillation of effective habit strength is, to a considerable extent, asynchronous.*

POSSIBLE SUBMOLAR CAUSES OF BEHAVIORAL OSCILLATION

There is reason to believe that one of the ultimate physiological or submolar causes of molar behavioral oscillation lies in the variability in the molecular constituents of the nervous system, the neurons. Blair and Erlanger have found, as the result of exceedingly delicate experiments on frog nerves, that neural response thresholds and reaction latencies of individual axon fibers vary spontaneously from instant to instant. They report:

When a preparation containing a fiber of outstanding irritability is stimulated with shocks increased in strength by small steps from below the fiber's threshold, there are at first only rare responses. To elicit a spike [electrical reaction] with every shock it usually is necessary to increase the strength further by about 2 per cent.

They report further:

The time intervening between successive threshold shocks and the resulting conducted axon spikes, or the shock-spike time, under exactly

comparable conditions is not constant. The range of fluctuation in the case of the more irritable fibers may be as great as 0.5σ , but usually is about 0.2 to 0.3σ ; in the case of the less irritable fibers it may be more than 2.4σ (1, pp. 530-531.)

If to the spontaneous oscillation in irritability of the neural conduction elements which mediate behavior, as reported by Blair and Erlanger, there be added the random and spontaneous firing of the individual neurons throughout the nervous system, which is indicated by the experimental evidence reported by Weiss (see p. 45, above), there would seem to be ample grounds for expecting oscillation to be a universal characteristic of organismic behavior (11).

The investigations just cited suggest not only the physiological cause of behavior oscillation but its characteristic distribution. Mathematicians have shown that the results of the joint action of a multitude of *independently* varying small factors tend to distribute themselves according to the so-called Gaussian or "normal law" of probability (2). Thus if 16 coins are tossed simultaneously a large number of times, and the number of heads coming up at each toss is recorded, it will be found that the most common number obtained will tend to be 8 and that the frequency of the other numbers of heads per throw tapers off symmetrically as zero and 16 heads per throw are approached. In a similar manner the spontaneous neural oscillations favoring a reaction stronger than average may be thought of on the analogy of the heads of the coins, whereas those favoring a reaction weaker than average may be considered analogous to the tails of the coins. Accordingly it is to be expected that in most cases the two opposing tendencies will be about equal in number and so will approximately balance each other, yielding a reaction of medium intensity. Occasionally, however, a disproportionate number of neural phases favoring strong or weak reactions will occur, just as a higher than average number of heads or tails in the coin-tossing experiment sometimes appears. On such occasions an unusually strong or an unusually weak reaction will be made, e.g., an unusually long or an unusually short line will be drawn. This, of course, agrees very well with the facts of gross behavior variability as represented in Figure 67. Other things equal, then, we may expect that *the magnitude of the contraction of each muscle involved in an act mediated by a receptor-effector connection will vary as a function of the normal law of probability.*

TABLE 9

THIS TABLE SHOWS (1) THE RELATIVE FREQUENCIES (IN PERCENTAGES) OF THE PROBABILITY INTEGRAL OVER EACH ONE-TENTH OF THE STANDARD DEVIATION, AND (2) THE CUMULATIVE PERCENTAGE VALUES FOR THE SAME INTEGRAL. THE DISTRIBUTION OF VALUES IS CUT OFF ARBITRARILY AT 2.5σ AT EACH SIDE OF THE CENTRAL TENDENCY. NOTE THAT ONE-HALF OF THE PROBABILITY DISTRIBUTION IS SHOWN IN COLUMNS b AND d , AND THAT THE OTHER HALF IS SHOWN IN COLUMNS b' AND d' . IT WILL BE OBSERVED THAT COLUMNS c AND d ARE DERIVED DIRECTLY FROM COLUMNS a AND b RESPECTIVELY. (Adapted from Thorndike, *g*.)

Deviations from the Central Tendency of Probability	Percentage of Probability Lying Between Each σ Entry and the One Preceding It in Column (a)	Deviations from a Point at -2.5σ from the Central Tendency of Probability	Cumulative Percentage of Probability Lying Between -2.5σ and the Entry in Column (a)	Deviations from the Central Tendency of Probability	Percentage of Probability Lying Between Each σ Entry and the One Preceding It in Column (a')	Deviations from a Point at -2.5σ from the Central Tendency of Probability	Cumulative Percentage of Probability Lying Between -2.5σ and the Entry in Column (a')
(a)	(b)	(c)	(d)	(e)	(a')	(e')	(d')
-2.5σ	.00	.0 σ	.00	.0 σ	.0 σ	2.5σ	49.37
-2.4σ	.20	.1 σ	.20	.1 σ	$+1.1\sigma$	2.6σ	53.35
-2.3σ	.25	.2 σ	.45	.2 σ	$+1.2\sigma$	2.7σ	57.29
-2.2σ	.32	.3 σ	.77	.3 σ	$+1.3\sigma$	2.8σ	61.16
-2.1σ	.40	.4 σ	1.17	.4 σ	$+1.4\sigma$	2.9σ	64.91
-2.0σ	.49	.6 σ	1.66	.6 σ	$+1.5\sigma$	3.0σ	68.51
-1.9σ	.60	.8 σ	2.26	.8 σ	$+1.6\sigma$	3.1σ	71.94
-1.8σ	.72	.7 σ	2.98	.7 σ	$+1.7\sigma$	3.2σ	75.17
-1.7σ	.86	.8 σ	3.84	.8 σ	$+1.8\sigma$	3.3σ	78.18
-1.6σ	1.02	.9 σ	4.86	.9 σ	$+1.9\sigma$	3.4σ	80.96
-1.5σ	1.20	1.0 σ	6.06	1.0 σ	$+2.0\sigma$	3.5σ	83.50
-1.4σ	1.39	1.1 σ	7.45	1.1 σ	$+2.1\sigma$	3.6σ	85.80
-1.3σ	1.60	1.2 σ	9.05	1.2 σ	$+2.2\sigma$	3.7σ	87.86
-1.2σ	1.83	1.3 σ	10.88	1.3 σ	$+2.3\sigma$	3.8σ	89.69
-1.1σ	2.06	1.4 σ	12.94	1.4 σ	$+2.4\sigma$	3.9σ	91.29
-1.0σ	2.30	1.5 σ	15.24	1.5 σ	$+2.5\sigma$	4.0σ	92.68
$-.9\sigma$	2.54	1.6 σ	17.78	1.6 σ	$+2.6\sigma$	4.1σ	93.88
$-.8\sigma$	2.78	1.7 σ	20.66	1.7 σ	$+2.7\sigma$	4.2σ	94.90
$-.7\sigma$	3.01	1.8 σ	23.87	1.8 σ	$+2.8\sigma$	4.3σ	95.76
$-.6\sigma$	3.23	1.9 σ	26.80	1.9 σ	$+2.9\sigma$	4.4σ	96.48
$-.5\sigma$	3.43	2.0 σ	30.23	2.0 σ	$+3.0\sigma$	4.5σ	97.08
$-.4\sigma$	3.60	2.1 σ	33.83	2.1 σ	$+3.1\sigma$	4.6σ	97.57
$-.3\sigma$	3.75	2.2 σ	37.58	2.2 σ	$+3.2\sigma$	4.7σ	97.97
$-.2\sigma$	3.87	2.3 σ	41.45	2.3 σ	$+3.3\sigma$	4.8σ	98.29
$-.1\sigma$	3.94	2.4 σ	45.39	2.4 σ	$+3.4\sigma$	4.9σ	98.54
$-.0\sigma$	3.98	2.5 σ	49.37	2.5 σ	$+3.5\sigma$	5.0σ	98.74
						5.1σ	100.00

SOME FURTHER CONSEQUENCES OF THE SIMULTANEOUS
FORTUITOUS VARIATION OF A VERY LARGE NUMBER
OF INDEPENDENT FACTORS

Mathematicians (2, p. 33 ff.) have determined by appropriate methods the outcome of what amounts to a coin-tossing experiment in which the number of coins is infinite and an infinite number of throws are made. The results of this mathematical procedure are conveniently presented in the form of a table, which is ex-

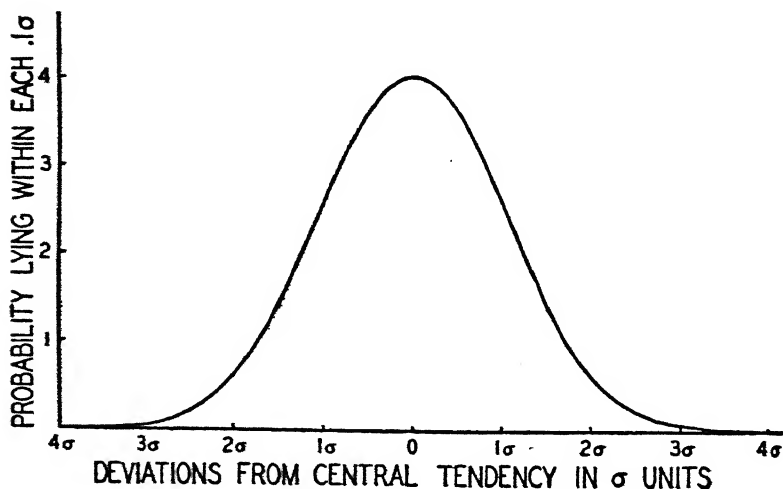


FIG. 69. Graphic representation of the distribution of normal probability to which the intensities of reactions evoked by repetitions of the same stimulus are believed to approach as a first approximation. This figure has been plotted mainly from columns a , b , a' , and b' of Table 9. Note the bell-shaped contour of the distribution.

tremely useful for reference, as it gives the standard form of distribution toward which all situations involving the action of numerous chance factors approach. An abbreviated adaptation of such an assemblage of theoretical chance values is shown in Table 9.

A graphical representation of one aspect of this particular phase of probability, that of the chance that the joint action of the factors will deviate by a given amount from the central tendency, is given in Figure 69. This figure was derived from Table 9 by plotting the values in columns b and b' as a function of the values in columns a and a' . Figure 69 should be compared with empirical Figure 67. It will be noted that Figure 69 has a smooth

contour, whereas Figure 67 varies by coarse steps; this difference is due to the infinite number of infinitesimal factors upon which Figure 69 is based.

The graphical representation of a second aspect of the joint action of an infinite number of small independent chance factors is given in Figure 70. This figure was also derived from Table 9, by plotting the values in columns d and d' as a function

of the values in columns c and c' . Figure 70 should be compared with the empirical graph shown in Figure 68. It will be noticed that Figure 68 resembles Figure 70 in that it is markedly sigmoid in shape; this fact strongly supports the hypothesis that a distribution of many independent chance factors produced the oscillation which occurred during the learning of the nonsense syllables, from the results of which Figure 68 was derived. Figure 68 differs markedly from Figure 70 in that the upper

portion of the S-shaped curve is much more extended than is the lower portion. This presumably is the result of the slower rate of habit strength acquisition as it approaches its physiological limit (see p. 116).

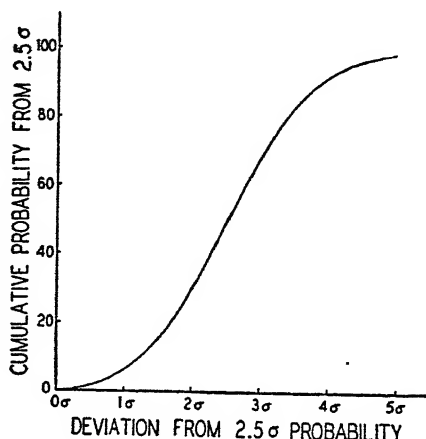


FIG. 70. Graphic representation of the cumulative per cent of normal probability from -2.5σ to $+2.5 \sigma$. This figure has been plotted from columns c , d , c' , and d' of Table 9. Because of its shape, this representation of the probability function is called the *ogive*.

THE MOLAR CONCEPTS OF BEHAVIORAL OSCILLATION (${}_sO_R$) AND OF MOMENTARY EFFECTIVE REACTION POTENTIAL (${}_s\bar{E}_R$)

At this point of our analysis we may formulate the molar concept of behavioral oscillation. On the basis of the submolar considerations presented above it is believed that the variability of reaction under seemingly constant conditions is due to the action of an oscillatory force upon the effective reaction potential (${}_s\bar{E}_R$). This oscillatory force will be represented by the symbol ${}_sO_R$. The momentary state of ${}_s\bar{E}_R$ under the influence of ${}_sO_R$ will be called

the *momentary effective reaction potential* and will be represented by the symbol $s\bar{E}_R$.

The evidence at present available indicates that sO_R rather closely approaches a normal chance distribution. A number of other critical matters in the situation are much less clearly evident. Among these latter problems is the question: Does the range of oscillation vary for a given organism, and if so upon what does this variability depend? This problem is particularly acute as the value of $s\bar{E}_R$ rises from zero to the reaction threshold. Closely related is the question of the direction of the momentary shift of $s\bar{E}_R$ under the influence of sO_R . For example, the action of sO_R might be wholly positive, causing $s\bar{E}_R$ always to oscillate upward; or its action might be distributed equally in both the positive and the negative direction. The lower portion of Figure 67 together with the conditions and facts of the acquisition of skill, which is believed to be closely related to the situation which yielded that distribution, suggests that $s\bar{E}_R$ may oscillate both upward and downward. On the other hand, the shape of certain curves of simple trial-and-error learning suggests that the action of sO_R on $s\bar{E}_R$ may be wholly negative and that its range may be substantially constant.

Partly as a means of facilitating exposition, but partly also as a means of opening the associated problems to much needed investigation, both empirical and theoretical, it has been decided to try out here the conceptually rather simple hypothesis suggested by the curve of simple trial-and-error learning, namely *that sO_R is an oscillating inhibitory potentiality, that it acts against effective reaction potential ($s\bar{E}_R$), that the distribution of sO_R conforms to the normal law of chance, that the mean value of sO_R and its range are both constant, and that the action of sO_R on the $s\bar{E}_R$ as applied to the several individual muscles is non-correlated.*

THE RESOLUTION OF THE "REACTION-EVOCATION" PARADOX

At this point we find ourselves in possession of principles adequate to explain the reaction-evocation paradox; i.e., how a stimulus may evoke a reaction which has never been conditioned either to it or to a stimulus in its stimulus-generalization range. The problem may, for convenience of exposition, be divided into two parts, (1) the resolution of the reaction-evocation paradox as ap-

plied to muscular contraction purely as such (to the so-called *actones* of Murray, 6, p. 54 ff.), and (2) the resolution of the same paradox as applied to *acts*, i.e., muscular contractions from the point of view of their effects upon the environment, particularly as these bear on the subsequent reinforcement of the movements in question.

The ultimate effector molar unit in habitual action is believed to be the individual muscle. This means not only that the action of each muscle in every coördinated movement must be mediated by a separate habit, but that every momentary phase of the contraction of every such muscle (since its proprioceptive cues are constantly changing) must be mediated by what is in some sense a different habit. Viewed in this manner, the contraction intensity of a given muscle, as mediated by the results of a given reinforcement, is the summation of the action of an uninterrupted chain or flux of habit, each phase of which is more or less distorted by the oscillation function. In this connection it must be recalled (p. 308) that the strength of each habit oscillates largely independently of all the others.

Now, nearly all movements are mediated by the coördination of sizable muscle groups. If the contraction of one muscle of such a group should vary in its intensity, that of the others remaining constant, the joint movement produced by the group as a whole will inevitably deviate in one respect or dimension from what it otherwise would have been. Since the contraction of each muscle is mediated by distinct habits, the contraction of all the muscles of a group will oscillate independently. Thus coördinated movement as such may be said to have as many dimensions of variation as there are muscles involved in its production. It follows from these considerations that infinitely varied movements other than those involved in the original conditioning process will inevitably be evoked by the impact of the conditioned stimulus, *S*. In this way the reaction-evocation paradox, from the point of view of movement as such, finds its resolution.

From the point of view of *action* as defined in the first paragraph of this section, it may be pointed out that behavioral oscillation gives rise to qualitative as well as quantitative differences. For example, if a particular muscle in a group mediating the striking of a typewriter key acts too weakly, the impression may be too faint to be legible; on the other hand, if some other muscle oscillates in the direction of too strong a contraction, the stroke may be

diverted to one side and a quite different key will be hit. In such a case a qualitatively different *act* or outcome may be said to have resulted from a quantitative deviation in an *actone* or movement.¹ Thus it is clear that very varied acts may result from the reinforcement of an extremely narrow zone of movements. In this way there emerges from the analysis the substance of what may be called *response intensity generalization*. Hence the reaction-evocation paradox, from the point of view of action and goal attainment, finds its resolution.

THE INFLUENCE OF THE OSCILLATION PRINCIPLE ON THE STATUS AND METHODOLOGIES OF THE BEHAVIOR SCIENCES

It is quite clear from the foregoing that the concrete manifestation of empirical laws (such as those concerning the acquisition of habit strength, p. 102 ff.) is bound to be greatly blurred by behavioral oscillation. Indeed, at first sight it might be thought that behavioral oscillation would preclude the possibility of any exact behavior science whatever. As a matter of fact, this pessimistic view is seriously held in certain quarters.

It must be confessed that behavioral oscillation does impose a grave handicap on all the social sciences; generally speaking, it precludes the possibility of deductively predicting the exact momentary behavior of single organisms. However, with an intimate knowledge of the history of the organism in question and a good understanding of the molar laws of behavior, it should be possible to predict *within the limits imposed by the oscillation factor* what the subject will do under given conditions. That behavior prediction has this limitation may be disappointing to some, particularly to individuals engaged in clinical practice, but there seems no escape from this difficulty; our task as scientists is to report what we find, rather than what we or our friends might wish the situation to be.

From the point of view of the general molar laws of behavior, the situation is far more satisfactory. Because of the tendency of large numbers of independent chance factors to distribute their influence more or less symmetrically (Figure 69) according to the Gaussian or normal law, it comes about that by various rather

¹ Fortunately, in most life situations reinforcement will follow about equally well movements possessing a considerable range of variability. Were this not so, organisms as now constituted could hardly survive.

simple statistical devices it is possible, when many comparable measurements have been made on the same individual's behavior or on that of a large number of comparable individuals, to isolate the central tendencies from the measures of individual reactions, more or less distorted as they are by the oscillation factor, and thus to reveal a close approximation to the laws which are operating. Mathematicians have shown that, other things constant, the distortions due to such chance factors vary inversely with the square root of the number of observations from which the central tendency is calculated. Thus the deviation of a mean calculated from 64 measures will in general differ from the "true" mean, i.e., that which might be calculated from an infinite number of comparable measures, by only half as much as that calculated from sixteen measures; the square root of 64 is 8, the square root of 16 is 4; and 4 is half as great as 8.

On the above principle it is evident that complete absence of blurring of the mean, due to oscillation and other chance irrelevant factors in the situation, is attained only when the number of measures becomes infinite; this means that *absolutely exact* empirical laws are never attainable. It follows that the most that can be hoped for in the empirical checking of the implications of behavioral laws must be greater or less degrees of approximation; and even this approximation can be attained only at the cost of great care and vast labor in the massing of data. Indeed, the universality of oscillation in organismic behavior is the main reason why the social sciences have been forced so extensively to employ statistical methods.

Finally, it may be said that the principle of behavioral oscillation is to a large extent responsible for the relatively backward condition of the social, as compared with the physical, sciences.

SUMMARY

Variability, inconsistency, and specific unpredictability of reaction under seemingly constant conditions are universal characteristics of the molar behavior of organisms, attested alike by general observation and by quantitative experiment. Neuro-physiological investigations suggest that behavioral oscillation arises from the spontaneously variable action of an enormous number of small factors (nerve cells), each acting independently to increase or decrease the intensity of reactions mediated by receptor-effector

connections. Where learned behavior is concerned, the oscillation is presumably of the effective reaction potentiality ($s\bar{E}_R$). In general confirmation of this view, typical experimental determinations show that with the effective habit strength and primary drive substantially constant, behavior evoked by successive repetitions of the same stimulus presents a close approximation to a normal probability curve. Moreover, evidence derived from trial-and-error learning situations demonstrates in a convincing manner that the oscillation associated with each habit tendency is largely, if not totally, uncorrelated with that of the others, i.e., that the oscillations of different effective habit tendencies are essentially asynchronous.

Additional indirect confirmation of the general Gaussian distribution of the oscillation function is found in the sigmoid shape of certain learning curves when plotted in terms of the per cent (or probability) of reaction evocation. The characteristically more protracted upper portion of these curves is presumably due in the main, at least, to the progressively slower rate of habit-strength acquisition as the physiological limit is approached.

Two important implications of the oscillation principle may be noted. The first yields an explanation for the superficial paradox that a stimulus is able to evoke reactions which are more or less distinct from any ever conditioned to it or to any other stimulus on the same stimulus continuum. The explanation lies in the tendency of the oscillation function to modify the intensity of every muscular contraction involved in every coördinated reaction; this makes the act evoked more or less different from any act involved in the original reinforcement. This amounts in effect to *response intensity generalization*.

A second implication of the principle of oscillation is that no theory of organismic behavior can ever be expected to mediate the precise prediction of the specific behavior of any organism at a given instant. However, because of the general regularity of the probability distribution in both its symmetrical and skewed forms, it will always be possible to predict approximately the central tendencies of behavior data from either individual organisms or groups of organisms which are under the influence of approximately the same antecedent factors and which share substantially the same neural and receptor-effector equipment. The oscillation factor explains why all of the behavior sciences derive their empirical laws from averages, and why their quantitative investigations necessi-

tate the securing of such great numbers of data. Finally, behavioral oscillation is believed to be, to a considerable extent, responsible for the present relatively backward state of the behavior (social) sciences.

On the basis of the considerations put forward in the preceding pages, we now formulate our tenth primary molar principle:

POSTULATE 10

Associated with every reaction potential (sE_R) there exists an inhibitory potentiality (sO_R) which oscillates in amount from instant to instant according to the normal "law" of chance. The amount of this inhibitory potentiality associated with the several habits of a given organism at a particular instant is uncorrelated, and the amount of diminution in sE_R from the action of sO_R is limited only by the amount of sE_R at the time available.

From Postulate 10 there follows Major Corollary III:

MAJOR COROLLARY III

Each muscular contraction involved in any increment of habit tendency (ΔsH_R) oscillates from instant to instant in the reaction-intensity potentiality which it mediates, thus producing a kind of response generalization in both directions from the response intensity originally reinforced.

NOTES

Mathematical Statement of Postulate 10

The mathematical statement of Postulate 10 is given by the following equation:

$$s\dot{E}_R = s\bar{E}_R - sO'_R, \quad (44)$$

where,

$s\dot{E}_R$ = the momentary effective strength of a reaction potential as modified by the oscillatory potentiality, sO_R ,

where,

$sO'_R = sO_R$ when $s\bar{E}_R \geq sO_R$,

and,

$sO'_R = s\bar{E}_R$ when $s\bar{E}_R < sO_R$,

zero $\leq sO_R \leq 6\sigma$, σ being a constant,

and,

the probability (p) that sO_R takes on values between zero and 6σ is p ,

where,

$$p = \frac{NS}{\sqrt{2}} e^{-\frac{(x-3\sigma)^2}{2\sigma^2}}$$

On the Oscillation of Effective Reaction Potentiality

A conception of behavior oscillation much like the one here elaborated was published by the author many years ago (4). Spearman (8, p. 323) incorporated it into his system of group factors which are supposed to determine human behavior, but otherwise the idea seems not, as yet, to have found acceptance or utilization. It would seem that as the theory of behavior grows more adequate and general, this principle in some form must find explicit recognition.

The equivalent of the principle of behavior oscillation appears as Postulate 15 of *Mathematico-Deductive Theory of Rote Learning* (5, p. 74). In that postulate, however, it was the reaction threshold which was supposed to oscillate, whereas in the present system it is reaction potential (sE_R) which is postulated as varying.

Finally, in the present system the principle of oscillation appears to be shifting from the status of a postulate or primary principle to that of a theorem or secondary principle. Such a change in the status of the principle of oscillation would be, of course, in accordance with the principle of parsimony, which is to the effect that, other things equal, the number of assumptions should be as few as possible.

The Derivation of Table 9 and Figures 69 and 70

Table 9 and Figures 69 and 70 are derived from the equation,

$$y = \frac{NS}{\sqrt{2\pi}} e^{-\frac{x^2}{2\sigma^2}}, \quad (45)$$

where N is the total number of chances (population) involved, y is approximately the number of these chances falling within a given interval, S is the extent of that interval in σ units, x is the distance of the midpoint of that interval from the central point of the distribution of chances, σ is the standard deviation of the distribution of chances, and π and e are mathematical constants with approximate values of 3.1416 and 2.718 respectively (7, p. 13).

The method of deriving columns b and b' of Table 9 (from which columns d and d' are derived) is illustrated by the following example:

Problem: To calculate the population of probability or chances falling within a range of $S = .1 \sigma$ (from $x - .05 \sigma$ to $x + .05 \sigma$) the midpoint of which is located at a distance, $x = 1.35 \sigma$, from the central tendency of the distribution of chances, the total population of chances being $N = 100$, the standard deviation of the distribution of chances being the unit of measurement of the range of the chances, i.e., $\sigma = 1$. Substituting these several values in the equation, we have,

$$\begin{aligned} y &= \frac{100 \times .1}{\sqrt{2 \times 3.1416}} \times 2.718^{\frac{-1.35^2}{2 \times 1^2}} \\ &= \frac{10}{\sqrt{6.2832}} \times 2.718^{\frac{-1.8225}{2}} \\ &= \frac{10}{2.506} \times 2.718^{-.91125} \\ &= 3.9893 \times .40248 \\ &= 1.6056, \end{aligned}$$

which agrees to the second decimal place with the entry in column *b*, opposite that of 1.3σ in column *a* in Table 9. This entry covers the range from 1.4σ to that of 1.3σ , the midpoint of which is 1.35, which is taken as the value of *x* in the above computations.

Behavioral Variability as Caused by the Introduction of Unaccustomed Components into the Conditioned Stimulus Compound

It is probable that an appreciable portion of the gross variability in the responses of organisms under approximately constant conditions of habit organization arises from the intrusion of a stimulus component, not present when the conditioning originally occurred, into the stimulus complex normally evoking the reaction. By the principle of afferent neural interaction (p. 42), such an intrusion would change to a certain extent the afferent impulse produced by the originally conditioned stimulus components. This, by the principle of the generalization gradient (p. 185), should weaken the resulting reaction, thus producing an oscillation in a downward direction. Numerous other variants in the stimulus would produce analogous quantitative variations in action evocation.

REFERENCES

1. BLAIR, E. A., and ERLANGER, J. A comparison of the characteristics of axons through their individual electric responses. *Amer. J. Physiol.*, 1933, 106, 524-564.
2. BROWN, W., and THOMSON, G. H. *The essentials of mental measurement*. London: Cambridge Univ. Press, 1921.
3. HILL, C. J. Retroactive inhibition in conditioned response learning. Ph.D. thesis, 1941, on file Yale Univ. Library.
4. HULL, C. L. The formation and retention of associations among the insane. *Amer. J. Psychol.*, 1917, 28, 419-435.
5. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
6. MURRAY, H. A. *Explorations in personality*. New York: Oxford Univ. Press, 1938.
7. RIETZ, H. L. *Handbook of mathematical statistics*. New York: Houghton Mifflin Co., 1924.
8. SPEARMAN, C. *The abilities of man*. New York: Macmillan Co., 1927.
9. THORNDIKE, E. L. *Theory of mental and social measurements*. New York: Teachers College, Columbia Univ., 1916.
10. THORNDIKE, E. L. *The fundamentals of learning*. New York: Teachers College, Columbia Univ., 1932.
11. WEISS, P. Functional properties of isolated spinal cord grafts in larval amphibians. *Proc. Soc. Exper. Biology and Medicine*, 1940, 44, 350 ff.

CHAPTER XVIII

The Reaction Threshold and Response Evocation

It will be recalled that more than once in the preceding pages, when discussing symbolic constructs such as sH_R , D , sE_R , \dot{I}_R , and $s\bar{E}_R$, we have emphasized the scientific hazards involved in their use. In this connection it has always been made clear that these dangers can be obviated only by having the constructs securely anchored in two temporal directions: (1) in objectively observable and measurable *antecedent* conditions or events, and (2) in objectively observable and measurable *consequent* conditions or events. Up to the present we have satisfied these requirements reasonably well in respect to the first or antecedent direction by laying down the conditions which culminate in effective reaction potential ($s\bar{E}_R$). To this end there have been shown in succession (1) the conditions, and (2) the relevant principles which generate habit strength (sH_R); which determine generalized habit strength ($s\bar{H}_R$); which show how drive (D) is generated, how habit strength and drive combine to produce reaction potential (sE_R), how inhibitory potentials (I_R and sI_R) are generated, how these combine with reaction potential to produce effective reaction potential ($s\bar{E}_R$), and how oscillation (sO_R) combines with $s\bar{E}_R$ to produce momentary effective reaction potential ($s\bar{E}_R$).

With the critical construct $s\bar{E}_R$ thus securely anchored on the antecedent side, we are at last free to consider the events and principles whereby it is anchored on the consequent side. In general the latter relationships are somewhat simpler than the former. Briefly stated, the consequent anchoring events are reactions (R), i.e., the movements or other activities of the organism. For the most part these reactions are susceptible of direct observation and automatic objective recording.

At present the clearest and most dependable single quantitative relationship subsisting between $s\bar{E}_R$ and R seems to be that of the probability (p) of the occurrence of the response following stimulation. Supplementing the probability-of-reaction-evocation relationship (p) are three additional functional relationships which materially contribute to the anchoring of the $s\bar{E}_R$ construct on the consequent side. These are: the latency of the reaction, the resist-

ance of the reaction potential to experimental extinction, and the amplitude of the reaction. In the delineation of the relationship of $s\bar{E}_R$ to R we shall accordingly take up first that based on the probability of reaction evocation. As a preliminary to this, however, it will be necessary to introduce the concept of the reaction threshold.

THE CONCEPT OF THE REACTION THRESHOLD (sL_R)

As used in neurophysiological, psychological, and behavior theory and empirical practice, the term *threshold* implies in general a quantum of resistance or inertia which must be overcome by an opposing force before the latter can pass over into action. So defined, the threshold concept fits many natural situations to which it is not customarily applied. Thus in beginning to drag a heavy object over a surface, one must often apply many pounds of traction before the weight begins to move perceptibly; if traction is gradually increased, there comes a point at which the addition of one more ounce to the pull starts the weight moving. The traction at this point would be the approximate threshold.

In an analogous manner, an appreciable weight must be placed on the skin before the subject can report its presence with a given degree of consistency; a certain amplitude of air vibration must reach the ear before the subject can consistently report a sound; a certain intensity of light must enter the eye before the subject can consistently report a color; the forearm must be moved a certain minimal number of units of arc at its joint before the subject can consistently report that passive movement has occurred. All of these are stimulus thresholds, traditionally supposed to be based primarily on the resistance or inertia of the receptor mechanisms.

In the early days of experimental psychology, as a result of the activities of Weber and Fechner (1), much time and energy were devoted to the determination of such thresholds. Because the early experimental psychologists were chiefly German and because of the prevalence of certain philosophical beliefs in Germany at the time, particularly those associated with metaphysical idealism, these minimal reportable stimulations were thought to represent a quantitative relationship between the physical and the psychic; this supposed transition of the physical into the psychic was thus conceived as a process of the physical stimulus entering the door of consciousness, hence the use of the word *threshold*. Accordingly,

the Latin word *limen* (threshold) is still commonly used as a synonym for the threshold in psychophysics. For this reason the threshold in psychology is represented by the symbol L ; this symbol, with the addition of a pair of qualifying subscripts, S and R , is employed in the present work to represent the reaction threshold, thus: ${}_SL_R$.

Proceeding to the matter of the quantitative operational definition of the threshold, it may be pointed out that in the chronaxie determinations of neurophysiology the threshold is defined as that minimal electrical current acting on any irritable tissue for an indefinite period which will evoke detectable activity, e.g., a discharge along a nerve fiber or a movement in a bit of muscle (2, p. 78 ff.). In an analogous manner, the reaction threshold (${}_SL_R$) is defined as the minimal effective reaction potential (${}_s\bar{E}_R$) which will evoke observable reaction; i.e., no reaction will occur unless

$${}_s\bar{E}_R - {}_SL_R$$

is greater than zero. This difference we shall call the *super-threshold effective reaction potential*.

INDIRECT DEMONSTRATIONS OF THE EMPIRICAL REACTION THRESHOLD

In ordinary behavior the reality of an empirical reaction threshold is demonstrated perhaps most clearly by the fact that in conditioning and other learning situations several reinforcements are frequently required before the stimulus will evoke the reaction. For example, in the conditioning of lid closure the conditioned reaction may be recorded as quite distinct from the blink which is associated with the air puff or whatever the reinforcing stimulus happens to be. It thus comes about that the question of whether or not the conditioned stimulus is able to evoke the reaction to which it is being conditioned, is readily determined empirically at all stages of the learning process without the usual complication of extinction effects. Utilizing this circumstance, Hill (5) found in one experiment that 15 per cent of his subjects showed their first conditioned reaction at the second reinforcing stimulation, 16.7 per cent at the third, 11.7 per cent at the fourth, 8.3 per cent at the fifth, 6.7 per cent at the sixth, and 3.3 per cent at the seventh stimulation. Other things equal, the number of reinforcements required before the first reaction evocation is a quantitative indication of

the height of the reaction threshold. Thus those subjects giving their first conditioned reaction at the sixth or seventh reinforcement presumably had higher empirical reaction thresholds than did those groups which gave their first conditioned reaction at the second or third reinforcement.

A rather different quantitative illustration of the empirical reaction threshold is presented in Figure 50 (p. 228). A careful examination of this figure shows that both fitted curves originate at the same point, namely, at a value of four extinction increments (ΔI_R) below the point at which response would be evoked, i.e., below the empirical reaction threshold. This seemingly paradoxical result is distinctly revealing as to the nature of the reaction threshold; since, as we have seen, reactions will not occur if the excitatory potential is below the reaction threshold, this extinction measure of effective excitatory potential cannot function when $s\bar{E}_R$ is less than sL_R . This means that the zero value of any *response* scale falls exactly at the reaction threshold.

Nevertheless we naturally wish to know how far the reaction threshold is above the absolute zero of effective reaction potential, or just no $s\bar{E}_R$ at all. Even though the extinction-reaction technique cannot directly enter this subthreshold region, it is possible to determine the shape of the learning function at numerous other points which are well above the reaction threshold, as shown for example by the circles in Figure 50. The curve or law of the function so determined, when extrapolated backward from the points empirically established to where $N = 0$ (and so, presumably, $s\bar{E}_R = 0$), yields an approximation to a value impossible of direct measurement. Thus the mean empirical reaction threshold of rats under the circumstances of Perin's experiment (8) purports to be an excitatory potential which would be approximately neutralized by the first four extinction reactions of the test series.

Before leaving this subject it must be pointed out that while the empirical reaction threshold includes the true or inertial threshold (sL_R), the two are not identical. There is good reason to believe (see sixth terminal note) that *not only the two types of empirical reaction thresholds just described, but all minimal stimulus thresholds in psychophysics are the sum of the true inertial threshold plus an artifact of undetermined magnitude which arises from the action of the oscillation function (sO_R)*. As yet no attempt has been made to determine the relative magnitude of the two factors entering into either the empirical reaction threshold or the

stimulus threshold of psychophysics; such a determination, while complicated and necessarily indirect, should be possible; when made it would not be surprising if the oscillation component is found to exceed in magnitude the true or inertial reaction threshold (sL_R). For the purposes of the present preliminary analysis, the quantitative separation of the two presumptive components is not necessary.¹

THE FUNCTIONAL RELATIONSHIP OF REACTION-EVOCATION
PROBABILITY (p) TO THE EFFECTIVE
REACTION POTENTIAL ($s\bar{E}_R$)

It will be recalled that as the result of certain considerations put forward in Chapter VIII (p. 102 ff.) we concluded that habit strength is a simple positive growth function of the number of reinforcements. Since reaction potential is a joint multiplicative function of habit strength and drive (p. 242), it follows that so long as drive remains constant, reaction potential will also closely approximate a simple growth function of the number of reinforcements. In the case of some types of response, such as salivary secretion and the galvanic skin reaction, the mean amplitude of response in simple learning situations has been found to be a positive growth function of the number of reinforcements (p. 103 ff.); this suggests a very simple (linear) relationship between the amplitude of such learned responses and effective reaction potential ($s\bar{E}_R$).

Many reactions, such as the bar-pressing movements of Perin's rats, approach the all-or-none type, which differs appreciably from the galvanic skin reaction, salivary secretion, etc. The all-or-none type of reaction introduces into the situation (1) the reaction threshold and (2) the oscillation of habit strength (p. 304 ff.). Owing to the simplicity of the threshold concept and to the fact that the oscillation function has already been fairly well established by independent investigations, the natural and strategic way to conceive the progress of learning from the response side is found in the probability (p) that the impact of the conditioned stimulus will evoke the response. Accordingly our examination of the quantitative relationship of effective reaction potential to the four modes of its observable manifestation in action will begin with the

¹ For further elaboration of this point, see sixth terminal note.

probability of the reaction evocation of the all-or-none type of response.

Let it be assumed that the habit strength of an all-or-none type of reaction is reinforced 36 times, with uniform time intervals between reinforcements great enough to prevent the accumulation of appreciable amounts of reactive inhibition (I_R). For the sake of simplicity in theoretical calculation, let it further be assumed that the drive is constant, that this and the conditions of reinforcement are such that the asymptote of effective reaction

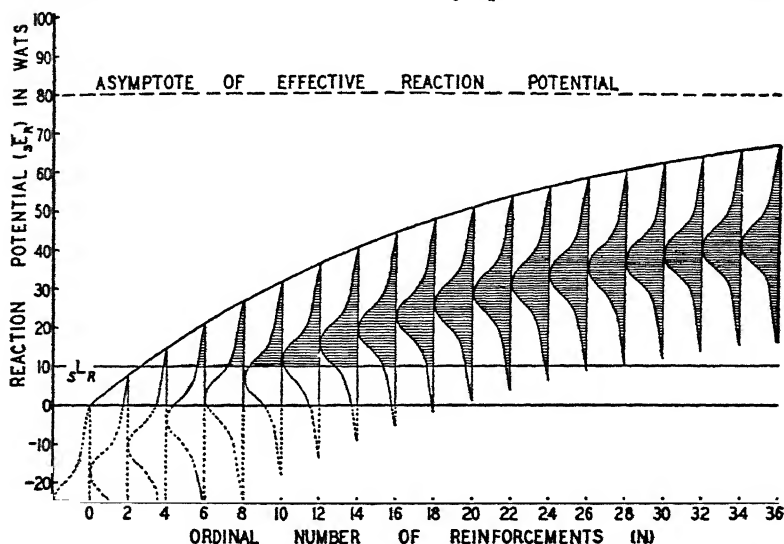


FIG. 71. Diagram showing the gradual movement of the zone of reaction-potential oscillation (up-ended bell-shaped areas) across the reaction threshold (sL_R). The upper or growth curve represents $s\bar{E}_R$ as a function of N and is plotted from columns 1 and 2 of Table 10. For further explanation see text.

potential ($s\bar{E}_R$) with unlimited practice will be 80 wats (p. 134 ff.), and that the nature of the reinforcing agent and related conditions surrounding the reinforcement process is such that the increment of effective reaction potential ($\Delta s\bar{E}_R$) at each reinforcement will be approximately one-twentieth of the difference between the effective reaction potential just preceding that reinforcement and the 80-wat asymptote. The reaction potentials just preceding each stimulation (and reinforcement) have been calculated on the above principle and are presented in numerical detail in the second column of Table 10. These values are represented graphically by the upward-arching growth curve shown in Figure 71.

Now, since reaction can be evoked only when the effective reaction potential exceeds the reaction threshold, i.e., when

$${}_s\overline{E}_R > {}_sL_R$$

and since by hypothesis in the present instance,

$${}_sL_R = 10 \text{ wats,}$$

it follows from the values in column 2 of Table 10 that the conditioned stimulations associated with the first three reinforcements cannot evoke the reaction being conditioned. Generalizing, we arrive at our first corollary:

I. *In the original learning of reactions of the all-or-none type, at least one and often a number of reinforcements are required before the reaction can be evoked by the conditioned stimulus alone, the number of reinforcements before reaction evocation being a decreasing function of the steepness of slope of the learning curve.*

It does not follow, however, that when

$${}_s\overline{E}_R > {}_sL_R$$

the impact of the conditioned stimulus will necessarily evoke the reaction being conditioned. This uncertainty comes from a number of independent considerations. The one which especially concerns us here is the oscillation principle, discussed at some length in an earlier chapter (p. 304 ff.). In order to illustrate the operation of the oscillation principle in the present situation, let it be assumed that the factors determining the oscillation of reaction potential are such that when operating at their *maximum* they are sufficient to neutralize any superthreshold effective reaction potential up to 50 wats which may be present, but when operating at a minimum, their neutralizing effect would be zero. Moreover, in accordance with considerations put forward in an earlier chapter (p. 308 ff.), the magnitude of these depressing effects presumably varies from moment to moment in a symmetrical manner about a central tendency according to the Gaussian "law" of probability. For convenience it will be assumed that the magnitude of oscillation varies over a range of 5σ (standard deviations); thus the standard deviation of oscillation (σ_0) in the supposed situation would have a value of 50 wats divided by 5, which yields a quotient of 10 wats.

With these values available and with the aid of a suitable table (based on the assumption of an infinite sample) we may determine the probability of reaction evocation after each reinforcement. The

TABLE 10

A TABLE SHOWING THE SEVERAL STEPS OF THE DERIVATION OF THE PROBABILITY OF REACTION EVOCATION AS A JOINT FUNCTION OF THE REACTION THRESHOLD, THE STRENGTH OF EFFECTIVE REACTION POTENTIAL ($s\bar{E}_R$), AND THE MAGNITUDE OF THE OSCILLATION OF THE REACTION POTENTIAL. STRICTLY SPEAKING, THESE VALUES, PARTICULARLY THOSE OF THE PROBABILITY FUNCTION (p), PRESUPPOSE AN UNLIMITED SAMPLE OF HOMOGENEOUS BEHAVIOR.

Number of Preceding Reinforce- ments (N) I	Effective Reaction Potential ($s\bar{E}_R$) II	Suprathreshold Effective Reaction Potential ($s\bar{E}_R - sL_R$) III	$(s\bar{E}_R - sL_R)$ σ_0 Where $\sigma_0 = 10$ IV	Probability of Reaction Evocation (p) Derived from Table 9 and Column IV V
0	0.00	.0	.0	.0
1	4.00	.0	.0	.0
2	7.80	.0	.0	.0
3	11.41	1.41	.141	.2
4	14.84	4.84	.484	1.66
5	18.10	8.10	.810	3.84
6	21.19	11.19	1.119	7.45
7	24.13	14.13	1.413	12.94
8	26.93	16.93	1.693	20.56
9	29.58	19.58	1.958	30.23
10	32.10	22.10	2.210	37.45
11	34.50	24.50	2.450	49.37
12	36.77	26.77	2.677	57.29
13	38.93	28.93	2.893	64.91
14	40.99	30.99	3.099	71.94
15	42.94	32.94	3.294	78.18
16	44.79	34.79	3.479	83.50
17	46.55	36.55	3.655	87.86
18	48.22	38.22	3.822	89.69
19	49.81	39.81	3.981	92.68
20	51.32	41.32	4.132	93.88
21	52.76	42.76	4.276	95.76
22	54.12	44.12	4.412	96.48
23	55.41	45.41	4.541	97.08
24	56.64	46.64	4.664	97.97
25	57.81	47.81	4.781	98.29
26	58.92	48.92	4.892	98.54
27	59.97	49.97	4.997	99.00
28	60.97	50.97	5.097	100.00
29	61.93	51.93	5.193	100.00
30	62.83	52.83	5.283	100.00
31	63.69	53.69	5.369	100.00
32	64.50	54.50	5.450	100.00
33	65.28	55.28	5.528	100.00
34	66.01	56.01	5.601	100.00
35	66.71	56.71	5.671	100.00
36	67.38	57.38	5.738	100.00

extent to which the upper limit of behavior oscillation exceeds the reaction threshold, i.e., the value of the superthreshold effective reaction potential ($s\bar{E}_R - sL_R$), is obtained by merely subtracting 10 from each of the $s\bar{E}_R$ values; these values are shown in column III of Table 10; they are indicated graphically in Figure 71 by the extent to which the up-ended, bell-shaped oscillation distributions project above the reaction threshold. This portion of the several distributions has been shaded to facilitate identification. The value of $s\bar{E}_R - sL_R$ is next divided by the value of the standard deviation of the oscillation (σ_o), i.e., by 10; the resulting ratios are given in column IV of Table 10.

Finally, the probability of reaction evocation at each reinforcement may be found by means of columns *c* and *d* of Table 9 (p. 311). For example, the *c*-value in Table 9 nearest to .141 is .1, for which the *d*, or probability, value is .2 per cent. Similarly, the *c*-value nearest to 1.119 is 1.1, which has a probability or *d*-value of 7.45 per cent, and so on. These probability values are given in column V of Table 10.

The moral of the rather complicated method of calculating *p* just described is that: *probability of reaction evocation is a normal probability (ogival) function of the superthreshold magnitude of effective reaction potential*. If this hypothesis, coupled with the growth hypothesis of the relation of the number of reinforcements to sH_R (and so to $s\bar{E}_R$), yields learning curves conforming substantially to those observed under corresponding empirical conditions, all hypotheses involved in the derivation will tend in so far to be substantiated.

THE REACTION-EVOCATION LEARNING CURVES IMPLIED BY THE THRESHOLD-OSCILLATION HYPOTHESIS

A graphic representation of the progressive movement across the reaction threshold of the zone of reaction-potential oscillations as a whole is shown in Figure 71. In each normal distribution the per cent that the shaded area stands to the entire area under the bell-shaped curve (column V of Table 10) is the probability (*p*) that an adequate stimulation will evoke the conditioned response. Because of their special significance these latter values are represented by a separate graph, which is shown in Figure 72. Actually this curve closely parallels an extensive group of empirical learning curves, a fact which strongly tends to substantiate the thresh-

old-oscillation hypothesis. A good example of this type of empirical learning curve from a simple conditioning situation is that of Hilgard and Marquis, shown in Figure 73. An example from a much more complicated experimental situation is given above, in Figure 68 (p. 307).

It is highly probable, however, that many learning curves of this general character reported in the literature have their shape determined in part at least by other factors. Presumptive examples of sigmoid learning curves so complicated are seen in Figure 24

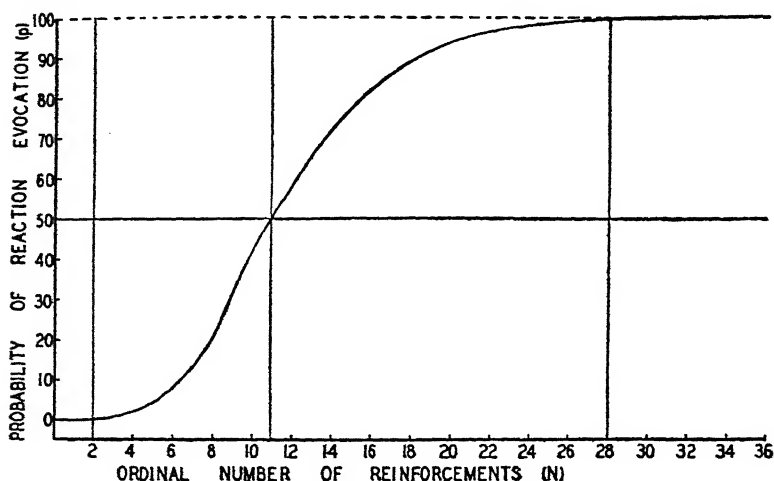


FIG. 72. Graph showing a theoretical probability-of-reaction-evocation curve of learning plotted as a function of the number of reinforcements. Note the somewhat distorted sigmoid shape of the learning curve as contrasted with the simple growth function shown in Figure 71. The extent of the distortion is indicated by the unequal separation of the three vertical lines drawn through the curve.

(p. 108). One of these complicating factors is the quasi-normal distribution in the learning difficulty of the various elements which are learned, e.g., the several syllables of rote series, the learning scores of which are pooled in the plotting of learning curves. Another source of the initial phase of positive acceleration sometimes found in learning curves is the interaction of the oscillation of two competing reaction potentials observed under certain conditions of simple trial-and-error learning (Figure 24 and p. 107 ff.).

It is quite evident that the characteristics of the curves of both Figure 72 and Figure 73 are radically different from those

of the simple positive growth function to which certain empirical learning curves approximately conform (see Figures 21 and 23, p. 103 ff.) and which we have found reason to believe also parallels the functional relationship of habit strength to the number of reinforcements. The relationships of the two contrasting types of learning curve may perhaps best be understood by comparing Figure 72 with the simple positive growth function from which it was derived; this is shown as the upper bounding curve in Figure 71. At a glance the probability-of-reaction-evocation curve is seen to be approximately sigmoid or ogival in form. A closer examination

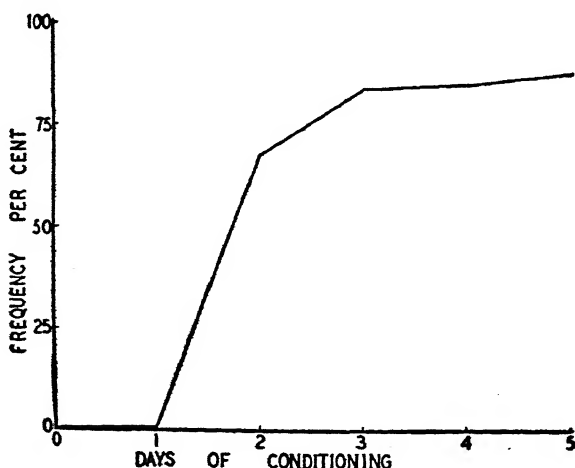


Fig. 73. An empirical probability-of-evocation type of learning curve showing a roughly sigmoid shape. This graph represents the frequency of conditioned lid reactions in dogs on each successive day of conditioning. (Adapted from Hilgard and Marquis, 4, p. 112.)

shows, however, that this learning curve differs from the true ogive (Figure 70, p. 313) in a number of important respects. In the first place, the probability-of-reaction-evocation function (p) has an initial section which is horizontal, standing at zero probability for three successive stimulations. This phenomenon has already been formulated as Corollary I. An empirical parallel to it may be observed in the initial portions of the curve shown in Figure 73.

A second and analogous difference lies in the fact that the probability-of-reaction-evocation curve has also a final phase of indefinite extent in which it is quite horizontal at 100 per cent, even though learning in the sense of increase in habit strength and

reaction potential progresses steadily (Table 10 and Figure 71). These considerations lead to the formulation of a second corollary:

II. *In the original simple conditioning or learning of an all-or-none type of reaction, the maximal level of 100 per cent of reaction evocation may occur in the later stages of reinforcement even though the reaction potential may steadily increase through continued reinforcement.*

It may be noted in connection with Corollaries I and II that the probability of reaction evocation (p) ceases to be an indicator of reaction potentiality both when the zone of oscillation is wholly below the reaction threshold and when it is wholly above it (Figure 71). For this reason, probability of reaction evocation in these extreme ranges is an entirely inadequate indicator either of habit strength or of effective reaction potential. Where reactions of the all-or-none variety are being learned, there are available, however, two supplementary measures throughout the considerable upper range of reaction potential which yields a uniform 100 per cent of reaction evocations. These are: (1) reaction latency (9), and (2) resistance to experimental extinction (11).

A further examination of Table 10 and Figure 72 shows that the probability-of-reaction-evocation learning curve differs from a true ogive (Figure 70) in that the steepness of rise of the first half is relatively greater than that of the second half. For example, the curve of Figure 72 rises from the last zero probability (after two reinforcements) to 50 per cent probability (after about 11 reinforcements) as the result of 11 — 2 or 9 reinforcements. On the other hand, it requires about 17 reinforcements (28 — 11) to pass from the 50 per cent level to the 100 per cent level. The true ogive of the normal probability function is, of course, quite symmetrical (see Figures 70 and 75). The asymmetry of the near-ogival probability-of-reaction-evocation learning curve is due to the influence of the progressively slower rise of the learning growth function ($s\bar{E}_R$) as it approaches its asymptote. Thus we arrive at our third corollary:

III. *The probability-of-reaction-evocation type of learning curve, while roughly resembling the normal ogive function, clearly deviates from it in that as the probability of reaction evocation increases from zero to 100 per cent, the rate of rise of the probability of reaction evocation is progressively slower as compared with the corresponding portion of the normal ogive.*

It is important to note in this connection that the initial period

of positive acceleration of the learning curve is to be expected on the present set of hypotheses only when the learning starts from absolute zero, or when the rate of learning is relatively slow, or when both these conditions obtain. Calculations have been made of the values of p when at the outset $s\bar{E}_R$ (possibly through generalization of excitation) is supposed to stand at 7 wats rather than at zero and when the fractional learning increment is taken at the relatively large value of $1/10$ rather than at $1/20$; these p values are represented graphically in Figure 74. There it may be seen that under the assumed conditions a fairly conventional growth-type curve of learning is to be expected, with scarcely any suggestion of the initial period of positive acceleration shown in Figure 72. It is believed that this is the explanation of the failure of many reactions of the all-or-none type to show the generally ogival form of learning curve. In this connection it may be recalled that Thur-

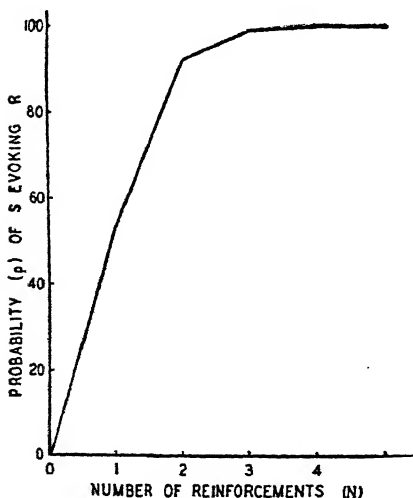


FIG. 74. A theoretical probability-of-reaction type of learning curve resulting from the assumption of a rapid rate of habit acquisition together with a substantial reaction potential from generalization at the outset of the reinforcements. See text for details.

stone found the sigmoid form of learning curve only when the material to be learned was difficult (10).

The preceding bit of analysis accordingly brings us to our fourth corollary:

IV. *If the learning of an all-or-none type of reaction sets out with an $s\bar{E}_R$ value appreciably above zero, and if the rate of learning is relatively rapid, the initial period of positive acceleration characteristic of this type of learning will not appear in the probability-of-reaction-evocation curve of learning.*

As a brief summary of the foregoing examination of the relationship of the theoretical construct, effective reaction potential ($s\bar{E}_R$), to reaction evocation as based on the threshold-oscillation hypothesis, we present in Figure 75, in comparable graphic form,

the two functions which combine to produce the sigmoid theoretical curve of learning shown in Figure 72. The upper portion of Figure 75 represents ${}_s\bar{E}_R$ as the familiar simple positive growth func-

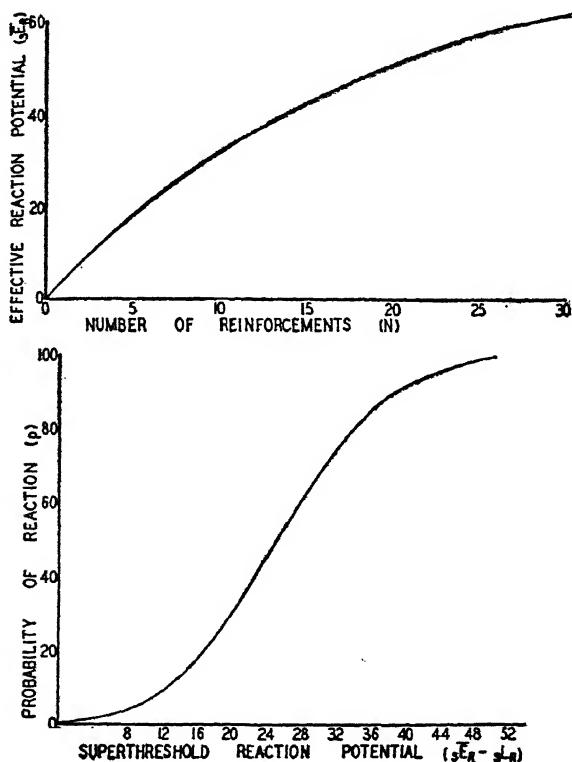


FIG. 75. Graph showing the analysis of the theoretical learning curve of Figure 72 into two components connected by the mediating construct ${}_s\bar{E}_R$. The upper curve shows ${}_s\bar{E}_R$ as a growth function of N , and the lower curve shows p as an ogival function of ${}_s\bar{E}_R - {}_sL_R$.

tion of the number of reinforcements (N). The lower portion of the figure shows the critical second component, the probability of reaction evocation, as an ogival function of ${}_s\bar{E}_R$, i.e., of the effective reaction potential less the reaction threshold. This latter type of function is the special concern of the present chapter, since it serves to anchor the construct ${}_s\bar{E}_R$ to an observable consequent event.

REACTION LATENCY (st_R) AS A FUNCTION OF EFFECTIVE REACTION POTENTIAL ($s\bar{E}_R$)

Proceeding with our systematic examination of the relationship of effective reaction potential to quantitatively observable response

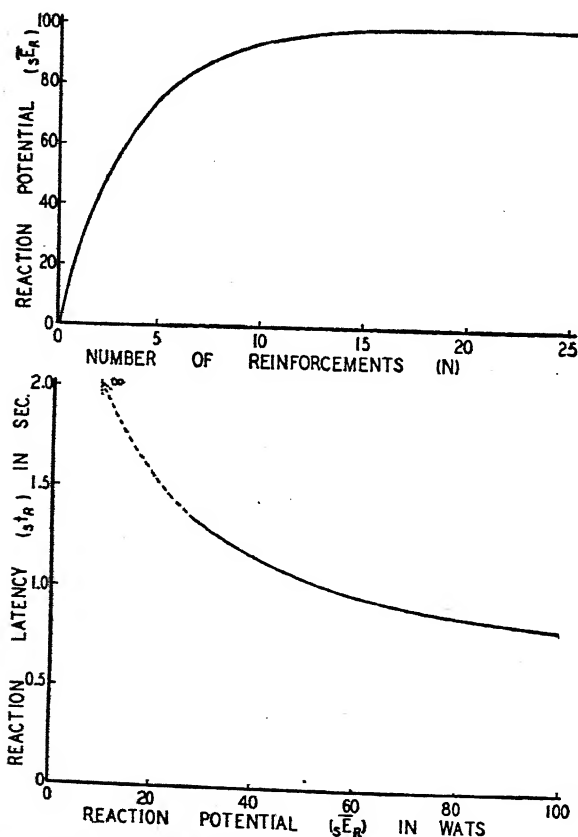


FIG. 76. Graphic representation of the theoretical components of one of Simley's empirical curves of learning (Figure 22) which is plotted in terms of reaction latency (st_R). The upper component is the familiar curve of habit strength (and so of $s\bar{E}_R$) plotted as a simple growth function of the number of reinforcements (N). The lower component represents st_R as a function of $s\bar{E}_R$. The broken line extending upward toward infinity as $s\bar{E}_R$ values grow less than about 24 wats represents an extrapolation into the region below the reaction threshold which in this case was attained after a single reinforcement.

phenomena, we consider next the functional relationship of $s\bar{E}_R$ to reaction latency, st_R , the time intervening between the begin-

ning of the stimulus and the beginning of the response. Unfortunately, the quantitative aspects of this function are complicated by the conditions of reinforcement in a manner not yet fully determined. It is well known, for example, that given suitable conditions of learning, organisms can be trained to react after a considerable range of predetermined delays. However, if the promptness of the reinforcement is dependent upon the promptness of the reaction, there is in general an inverse functional relationship of the number of reinforcements to the reaction latency. The following analysis assumes these latter learning conditions.

This relationship is perhaps best represented by means of a graphic analysis of the Simley (9) reaction-latency learning curve shown in Figure 22. Such an analysis is presented in Figure 76 in a manner parallel to that of the purely theoretical probability-of-evocation function shown in Figure 75. Thus in the upper portion of Figure 76 there appears the familiar positive growth function representing the relationship of the effective reaction potential to the number of reinforcements. In the lower portion of the figure we note the functional relationship which is of present interest, that of st_R to $s\bar{E}_R$. A glance at this portion of the figure shows that st_R is a negatively accelerated decreasing function of $s\bar{E}_R$ where $s\bar{E}_R$ is greater than about 24 wats, the empirical reaction threshold.

RESISTANCE TO EXPERIMENTAL EXTINCTION (n) AS A FUNCTION OF EFFECTIVE REACTION POTENTIAL ($s\bar{E}_R$)

Continuing our systematic examination of the relationship of effective reaction potential to quantitatively observable response phenomena, we attempt as our third task the determination of the functional relationship of $s\bar{E}_R$ to the number of unreinforced reaction evocations (n) required to extinguish a reaction potential to a given degree of impotence, say to three successive stimulations which fail to evoke observable reaction. Here, much as in the case of reaction latency, the quantitative aspects of the problem are complicated by the conditions of reinforcement. It has been shown by Humphreys (7), for example, that the course of extinction is rather different when the reinforcements of the original learning have been accompanied by a considerable number of non-reinforced stimulations. The present analysis is accordingly somewhat tentative, as was that concerned with st_R . It proceeds on the assump-

tion that the conditions of reinforcement are constant throughout and that they are uncomplicated by non-reinforcement or other disturbing factors.

This relationship may perhaps best be illustrated by means of

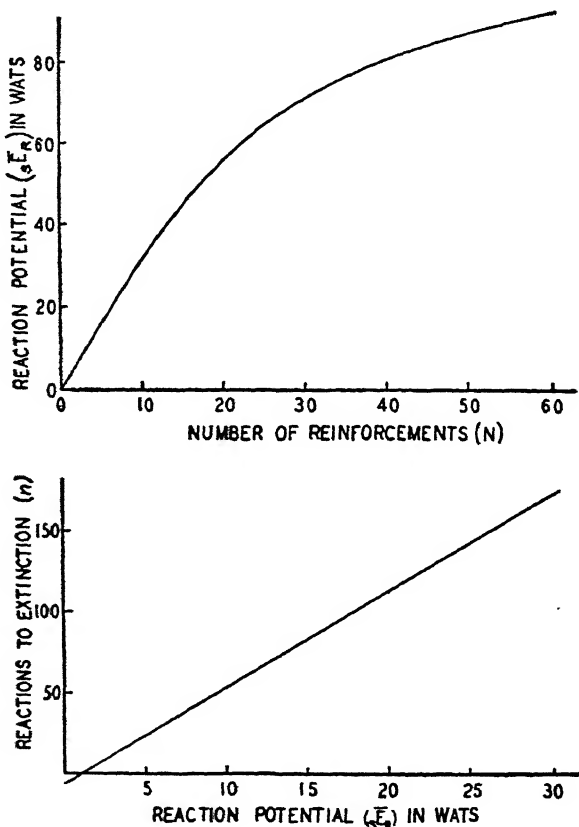


FIG. 77. Graphic representation of the theoretical components of Williams' empirical learning curve (Figure 23) which is plotted in terms of the number of unreinforced reaction evocations (n) required to produce experimental extinction. The upper component is the usual curve of habit strength (and so of E_R) plotted as a simple growth function of the number of reinforcements (N). The lower component represents n as a simple linear function of E_R .

a graphic analysis of Williams' learning curve shown in Figure 23. This curve, it will be recalled, represents the number of unreinforced reactions (n) required to produce a given degree of extinction as a function of the number of reinforcements, drive remaining

constant. The theoretical analysis of this empirical function into two components in parallel with the analysis presented in Figures 75 and 76, is shown in Figure 77. As in Figures 75 and 76, the upper portion of this figure represents reaction potential as the familiar positive growth function of the number of reinforcements. The lower curve shows as the second component that *the number of extinction reactions (n) is a simple increasing linear function of the reaction potential (${}_s\bar{E}_R$).*

It may be noted incidentally that, according to this function, n becomes negative when ${}_s\bar{E}_R$ has values less than about 8 wats. This, of course, is an expression of the reaction threshold emphasized in connection with the interpretation of Figure 50.

INTENSITY OR AMPLITUDE OF REACTION (A) AS A FUNCTION OF EFFECTIVE REACTION POTENTIAL (${}_s\bar{E}_R$)

As our fourth and concluding analysis of the relationship of effective reaction potential to quantitatively observable response phenomena, we shall consider that of ${}_s\bar{E}_R$ to a variety of reaction which is *not* of the all-or-none type. We have chosen for this purpose Hovland's empirical curve of the acquisition of a conditioned galvanic skin reaction, shown as Figure 21 (p. 103). In connection with this analysis it must be pointed out, much as in the cases of reaction latency and of extinction, that the function may possibly be complicated by variations in the conditions of the original acquisition of the reaction potential. It is known (p. 305 ff.) that striated-muscle reaction may easily be trained to a particular amplitude by special conditions of reinforcement. However, since no evidence, experimental or observational, has been found of such a tendency in the case of either the galvanic skin reaction or salivary secretion, distorting complications of the functional relationship of A to ${}_s\bar{E}_R$, where the autonomic nervous system is primarily involved, seem rather unlikely. At all events, it is assumed in the following analysis that no such complications are involved.

The components into which this learning curve breaks up are presented in Figure 78 in a manner exactly parallel to Figures 75, 76, and 77. The upper portion of Figure 78 shows, as usual, the effective reaction potential (${}_s\bar{E}_R$) as a simple positive growth function of the number of reinforcements (N). The lower portion of the figure, our chief concern here, reveals that *the amplitude of the conditioned galvanic skin reaction is a simple linear increasing*

function of the effective reaction potential. It thus resembles very closely the relationship of $s\bar{E}_R$ to the number of unreinforced reaction evocations (n) required to produce extinction. However, a striking difference is also to be noted: whereas in Figure 77 the straight line originates below the reaction threshold, in Figure 78 it originates an appreciable distance above it; this reflects the

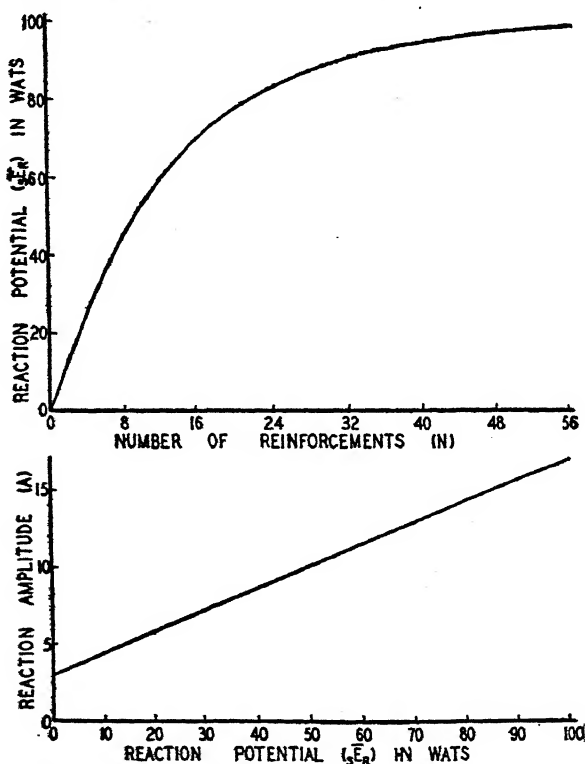


FIG. 78. The analysis of a curve of the conditioning of the galvanic skin reaction (Figure 21) into two components connected by the theoretical construct $s\bar{E}_R$.

well-known fact that previous to specific conditioning, almost any stimulus will evoke the galvanic skin reaction. Stated in another way, this means that at the outset of the learning process here under consideration (Figure 21), the reaction tendency is well above the reaction threshold, just as that shown in Figure 50 is appreciably below it.

THE COMPETITION OF SIMULTANEOUS INCOMPATIBLE REACTION POTENTIALS

There remains to be considered one more important matter before the relationship between $s\bar{E}_R$ and reaction is formally complete. It will be recalled that when discussing the reaction threshold we pointed out above that no reaction will be evoked unless $s\bar{E}_R$ is greater than sL_R . It must now be noted that reaction will not inevitably occur when $s\bar{E}_R$ exceeds sL_R , or even when the momentary effective reaction potential ($s\bar{E}_R$) is greater than sL_R . This is because *there are frequently encountered situations in which the stimulus complex impinging on the organism may simultaneously give rise to two or more incompatible reaction potentials*. Examples of such reaction tendencies would be opening and closing the eyelids, extending and flexing the arm or leg, or speaking almost any two words of a language. It is obvious that in such a situation the momentarily weaker of two competing reaction potentials cannot possibly mediate its reaction.

Whether in such cases the reaction potential of the dominant tendency is completely brought to bear in the evocation of the reaction, or whether it suffers some diminution resembling that long known as *associative inhibition*, is not known. Certain observations suggest that the latter supposition is the true one. At all events, the experimental evidence has led some investigators (3, p. 206) to the view that the interference of incompatible reaction potentials may be mutual and that this generates an inhibitory potential which behaves in many, if not all, respects as does that generated by experimental extinction. Unfortunately the dynamics of this very common situation have not been sufficiently investigated to warrant an attempt at a detailed quantitative statement, particularly as to possible indirect inhibitory effects.

Ignoring for the present, then, possible generalized inhibitory tendencies which may result from the competition of incompatible reaction potentials, we state as a first approximation that *if two or more superthreshold reaction potentials exist in an organism at the same instant, only the reaction of that one whose oscillation value at the moment is greatest will be evoked*. Thus is formally concluded the task of anchoring the construct $s\bar{E}_R$ to objectively observable behavior on the consequent side.

SUMMARY

The pivotal theoretical construct of the present system is that of the effective reaction potential ($s\bar{E}_R$). An attempt has been made in the preceding chapters to anchor this in a secure and quantitative manner to antecedent observable conditions of habit formation, of motivation, and of stimulation immediately preceding reaction evocation. The task of the present chapter has been to anchor it in a parallel manner on the posterior or consequent side. The observable phenomena available for this purpose are found in four aspects of reaction evocation: (1) the probability (p) of reaction evocation; (2) reaction latency (st_R); (3) resistance to experimental extinction (n); and (4) in the case of autonomically mediated reactions, reaction amplitude (A). This means that a successful performance of our task involves the quantitative determination of the functional relationship of $s\bar{E}_R$ to p , st_R , n , and A , respectively.

Of the four relationships, the one at present offering the best prospects of a successful conclusion is that involving the probability of reaction evocation, p . This is in part because the various additional constructs necessarily involved are revealed in a fairly obvious manner by relatively independent considerations. The constructs in question are (1) the reaction threshold (sL_R) and (2) the downward oscillation (sO_R) to which effective reaction potential ($s\bar{E}_R$) is believed to be subject.

So long as the maximum reaction potential lies below the reaction threshold, no activity can be evoked. However, as this maximum passes the reaction threshold in the more simple learning situations, the probability of reaction evocation increases progressively until the zone of oscillation is wholly above the reaction threshold, when the probability of reaction will be 100 per cent, or perfect. Since it may require several reinforcements to raise $s\bar{E}_R$ to a value exceeding that of sL_R , it often happens that there is an initial region of uniformly zero reaction probability in learning curves. Similarly, the zone of maximum oscillatory interference with $s\bar{E}_R$ (minimum value of $s\bar{E}_R$) may have passed above the reaction threshold before the limit of habit acquisition afforded by the conditions of reinforcement is reached; this may bring about a more or less protracted terminal period of uniformly perfect response probability (100 per cent) in this type of learning curve. Finally, owing to the presumably normal distribution of the oscil-

lation function, its passage across the reaction threshold yields during the learning process a probability-of-reaction-evocation learning curve which possesses when complete a distinctly skewed ogival form. The general agreement of learning curves secured under corresponding empirical conditions, with the three theoretical implications just listed, gives considerable additional substantiation to the belief in the general soundness of the various hypotheses involved.

One of the results emerging from the above analysis is a further confirmation of the critical hypothesis that sH_R (and so $s\bar{E}_R$, in case D is constant) is a simple positive growth function of the number of reinforcements (N). This last consideration is of strategic importance because it enables us to determine in an indirect manner the presumptive functional relationship of each of the three remaining response phenomena to the standard or maximal value of effective reaction potential ($s\bar{E}_R$). It happens that the sample empirical learning curve based on each of the three response aspects is expressible to a reasonably close approximation by equations, all of which contain an explicit positive growth function of N . The replacement of this expression by its equivalent, $s\bar{E}_R$, leaves the particular response phenomenon stated in terms of $s\bar{E}_R$. This type of analysis when applied to the learning curves based on presumably representative sets of empirical data indicates as a first approximation that: reaction latency (st_R) is a negatively accelerated decreasing function of effective reaction potential ($s\bar{E}_R$); that resistance to experimental extinction (n) is an increasing linear function of effective reaction potential ($s\bar{E}_R$); and that amplitude of reaction (mediated by the autonomic nervous system) is an increasing linear function of effective reaction potential ($s\bar{E}_R$).

However, situations frequently occur where the stimulus complex impinging on the organism at a given moment is such as to give rise to two or more incompatible reaction potentials. Ignoring for the present the possibility of indirect inhibitory effects, we may say as a first approximation that in such situations the reaction potential which is strongest will dominate the others by evoking its reaction. Thus is formally completed the anchoring of the constructs $s\bar{E}_R$ and $s\bar{\bar{E}}_R$ to objectively observable phenomena on the consequent side.

Generalizing on the considerations elaborated above, we now formulate six additional primary principles:

POSTULATE 11

The momentary effective reaction potential ($s\dot{E}_R$) must exceed the reaction threshold (sL_R) before a stimulus (S) will evoke a given reaction (R).

POSTULATE 12

Other things equal, the probability (p) of striated-muscle reaction evocation is a normal probability (ogival) function of the extent to which the effective reaction potential ($s\dot{E}_R$) exceeds the reaction threshold (sL_R).

POSTULATE 13

Other things equal, the latency (st_R) of a stimulus evoking a striated-muscle reaction is a negatively accelerated decreasing monotonic function of the momentary effective reaction potential ($s\dot{E}_R$), provided the latter exceeds the reaction threshold (sL_R).

POSTULATE 14

Other things equal, the mean number of unreinforced striated-muscle reaction evocations (n) required to produce experimental extinction is a simple linear increasing function of the effective reaction potential ($s\dot{E}_R$) provided the latter at the outset exceeds the reaction threshold (sL_R).

POSTULATE 15

Other things equal, the amplitude (A) of responses mediated by the autonomic nervous system is a simple linear increasing function of the momentary effective reaction potential ($s\dot{E}_R$).

POSTULATE 16

When the reaction potentials (sE_R) to two or more incompatible reactions (R) occur in an organism at the same time, only the reaction whose momentary effective reaction potential ($s\dot{E}_R$) is greatest will be evoked.

Postulate 16 completes our statement of primary principles.

NOTES

Mathematical Statement of Postulate 11

$$(Sx) \cdot x \in S \cdot s\dot{E}_R > sL_R \supset (Ry) \cdot y \in R \quad (46)$$

Mathematical Statement of Postulate 12

$$\begin{aligned}
 &= 0 \text{ when } s\bar{E}_R \leq sL_R \\
 &= \frac{81}{80} \left[\psi(2.5) - \psi\left(\frac{sL_R - s\bar{E}_R + 2.5\sigma}{\sigma}\right) \right] \\
 &= 1 \text{ when } s\bar{E}_R \geq sL_R + 5\sigma,
 \end{aligned} \tag{47}$$

where σ is the standard deviation of sO_R ,
and

$$\psi(\sigma) = \int_{-\infty}^{\sigma} \phi(t) dt,$$

where $\phi(t)$ is the standard probability function.

Mathematical Statement of Postulate 13

$$s\bar{t}_R = \frac{a'}{s\bar{E}_R^{b'}}, \tag{48}$$

where a' and b' are positive empirical constants.

Mathematical Statement of Postulate 14

$$n = c' s\bar{E}_R - f', \tag{49}$$

where c' and f' are empirical constants.

Mathematical Statement of Postulate 15

$$A = h' s\bar{E}_R - i', \tag{50}$$

where h' and i' are empirical constants.

Mathematical Statement of Postulate 16

$$(Ax) \cdot w \in s\bar{E}_{R_1} : (Ax) \cdot x \in s\bar{E}_{R_2} : (Ay) \cdot y \in S : s\bar{E}_{R_1} > s\bar{E}_{R_2} > sL_R : \supset : (Ax) \cdot z \in R_1 \tag{51}$$

The Oscillation Component of the Empirical Reaction Threshold

This component would arise in a simple reinforcement determination of the reaction threshold (as in the experiment cited from Hill, 5) in the following manner: As the (maximum) value of $s\bar{E}_R$ is passing the reaction threshold, it is in the highest degree improbable that $s\bar{E}_R$ will be depressed to the minimal (zero or near-zero) degree of oscillation the first time $s\bar{E}_R$ is tested for reaction failure. For example, on the assumption that the maximum range of oscillation is 5σ , the chances are 95 to 5 against $s\bar{E}_R$ exceeding the reaction threshold even when the standard or maximal value of $s\bar{E}_R$ has risen above the reaction threshold by as much as one-fifth its range of oscillation, i.e., nearly as much as that shown at $N = 6$ in Figure 71. This means, of course, that with a limited number of trials at this level of learning there will inevitably be an appreciable mean value of $s\bar{E}_R$ previous to the first reaction evocation. It is evident that the extent to which this mean value of $s\bar{E}_R$ at the first reaction evocation exceeds zero must be

included in the magnitude of the empirical reaction threshold as indicated above from conditioning data. Indeed, it would produce a considerable superficial indication of an empirical reaction threshold even though no inertial reaction threshold (sL_R) were present.

By exactly analogous reasoning it is easy to show that a parallel effect would result from the determination of the empirical reaction threshold by the pooling of results secured in the ordinary extinction of all-or-none reactions.

The Equations From Which the Curves in Figure 75 Were Plotted

The equation of the upper or growth function is,

$$s\bar{E}_R = 100 (1 - 10^{-.02226 N}).$$

The equation expressing the functional dependence of p upon $s\bar{E}_R$ is,

$$\begin{cases} = 0 & \text{if } s\bar{E}_R \leq sL_R \\ = 1.0125 [\psi(2.5) - \psi(3.5 - .1s\bar{E}_R)] & \\ = 1 & \text{if } s\bar{E}_R \geq sL_R + 5\sigma, \end{cases}$$

where σ is the standard deviation of the oscillation function (sO_R), sL_R is taken as 10 wats, and 5σ is taken as 50 wats. *This is the first of the four critical equations anchoring the construct $s\bar{E}_R$ on the consequent side to objectively observable phenomena.* The second of the three right-hand members of the above expression represents the normal ogive function.

The Equations From Which the Curves in Figure 76 Were Plotted

The equation fitted to one of Simley's composite empirical learning curves (Figure 22 plotted in terms of reaction latency) (sL_R) is,

$$sL_R = \frac{5.11}{[100 (1 - 10^{-.122N})]^4}.$$

Inspection of the right-hand member of this equation reveals the expression $(100(1 - 10^{-.122N}))$ which is the familiar expression of $s\bar{E}_R$ as a positive growth function of N . This circumstance enables us directly to write the equation,

$$s\bar{E}_R = 100 (1 - 10^{-.122N}),$$

from which was plotted the curve in the upper portion of Figure 76.

Replacing $100 (1 - 10^{-.122N})$ in the first equation by $s\bar{E}_R$, we have as the other component,

$$sL_R = \frac{5.11}{s\bar{E}_R^4},$$

from which was plotted the lower curve of Figure 76. *This is the second of the four critical equations anchoring the construct $s\bar{E}_R$ on the consequent side to objectively observable phenomena.* It should be noted that, strictly speaking, this equation does not hold for $s\bar{E}_R$ values below about 24 wats, which in this case marks the reaction threshold.

The Equations From Which the Curves of Figure 77 Were Plotted

The equation fitted to Williams' and Perin's empirical learning curve (Figure 23) is,

$$n = .66 [100 (1 - 10^{-.018N})] - 4.$$

By inspection, the bracket in the right-hand member of this equation is a positive growth function of N and presumably represents $s\bar{E}_R$. We accordingly may write the equation,

$$s\bar{E}_R = 100 (1 - 10^{-.018N}).$$

It is from this equation that the upper curve of Figure 77 is plotted.

Replacing the bracket of the original fitted equation by $s\bar{E}_R$, we may write from equation 43,

$$n = \frac{(B - W)(s\bar{E}_R - sL_R)}{c},$$

where $B = 116.6$, $W = 42.5$, $c = 12.5$, and $L = 1$, as the remaining component of the empirical equation. It is from this equation that the lower curve of Figure 77 has been plotted. *This is the third of the four equations anchoring the critical construct, $s\bar{E}_R$, on the consequent side to objectively observable phenomena.*

It should be noted in this connection that certain evidence, such as that represented by Figure 57 and Table 6, appears superficially to be in conflict with the linear relationship of n as a function of $s\bar{E}_R$, represented by the above equation and in Figure 77. It is possible that the apparent disagreement is due to the fact that both Table 6 and Figure 57 are based on processes under autonomic control, whereas Figure 77 presupposes striated muscle response. Because of the seeming inconsistency in the evidence, Postulate 14 and the above equation expressing it cannot be accepted without reservation until definite confirmatory evidence becomes available.

The Equations From Which the Curves of Figure 78 Were Plotted

The equation fitted to Hovland's empirical learning curve data (Figure 21) is,

$$A' = .141 [100 (1 - 10^{-.033N})] + 3.1.$$

By inspection, the bracket may be seen to enclose a simple positive growth function of N . Accordingly we write directly the equation, -

$$s\bar{E}_R = 100 (1 - 10^{-.033N}).$$

Replacing the bracket of the original equation by $s\bar{E}_R$, we have as the second component,

$$A = .141 s\bar{E}_R + 3.1.$$

It is from this that the lower curve of Figure 87 has been plotted. *This is the last of the four equations anchoring the critical construct $s\bar{E}_R$ on the consequent side to objectively observable phenomena.*

REFERENCES

1. BORING, E. G. *A history of experimental psychology*. New York: Century Co., 1929. Now published by D. Appleton-Century Co., Inc.
2. FULTON, J. F. *Muscular contraction and the reflex control of movement*. Baltimore: Williams and Wilkins Co., 1926.
3. GIBSON, E. J. A systematic application of the concepts of generalization and differentiation to verbal learning. *Psychol. Rev.*, 1940, 47, 196-229.
4. HILGARD, E. R., and MARQUIS, D. G. *Conditioning and learning*. New York: Appleton-Century Co., Inc., 1940.
5. HILL, C. J. Retroactive inhibition in conditioned response learning. Ph.D. thesis, 1941, Yale University.
6. HOVLAND, C. I. The generalization of conditioned responses. IV. The effects of varying amounts of reinforcement upon the degree of generalization of conditioned responses. *J. Exper. Psychol.*, 1937, 21, 261-276.
7. HUMPHREYS, L. G. The effect of random alternation of reinforcement on the acquisition and extinction of conditioned eyelid reactions. *J. Exper. Psychol.*, 1939, 25, 141-158.
8. PERIN, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. *J. Exper. Psychol.*, 1942, 30, 93-113.
9. SIMLEY, O. A. The relation of subliminal to supraliminal learning. *Arch. Psychol.*, 1933, No. 146.
10. THURSTONE, L. L. The learning curve equation. *Psychol. Monog.*, 1919, 26, No. 114.
11. WILLIAMS, S. B. Resistance to extinction as a function of the number of reinforcements. *J. Exper. Psychol.*, 1939, 23, 506-521.

CHAPTER XIX

The Patterning of Stimulus Compounds

In our account of the "law of reinforcement" (p. 71 ff.) it was pointed out with care that reactions are conditioned to the central afferent impulses (s) set in motion by the action of stimulus energies (S) upon receptors. Because of the approximate one-to-one relationship between S and s , in many instances the influence of the principle of afferent neural interaction (p. 42 ff.) was largely ignored in the interest of expository simplicity. For similar reasons the distinction between sH_R and sH_R was not stressed, and the habit strengths to the evocation of a given reaction associated with different stimulus elements or aggregates occurring together were reported in the main, though not exclusively (p. 216 ff.), as summing by a kind of diminishing returns principle (p. 223 ff.). Moreover, a detailed account of the rôle of afferent neural interaction, particularly in the patterning of stimulus compounds, could not be given until the reader had been familiarized with the major phenomena of conditioned inhibition (p. 282 ff.), of its generalization (p. 283 ff.), and of oscillation (p. 304 ff.). Now that all of our primary principles have been set forth, we may present in a little detail some further important implications of the principle of afferent neural interaction.

For reasons presently to be disclosed (p. 374) there are many situations in which organisms would have about as good chance of survival if they responded as if their reactions were conditioned to stimulus elements (S) as they would if their reactions were conditioned to central afferent impulses (s); moreover, habits do in fact summate (p. 209 ff.). There are, however, innumerable situations in which the response *must* be made to a certain combination or configuration of circumstances (2, p. 503) and in which that response would not be reinforced if given to any single circumstance as represented by isolated stimulus elements or aggregates¹ or in any combination. For example, a red light suspended

¹ Actually, of course, stimulus elements probably never literally occur alone; a stimulus element or aggregate is said to occur "alone" when its onset occurs alone, the onset of the other stimuli having occurred earlier or later. A stimulus element is a stimulus energy which activates a single re-

over a street intersection will cause a man to halt when his goal would lead him to cross the street, but a red light in a drugstore window will not cause him even to slow his pace; he responds not to the red light alone, but to it as a component in a particular combination of stimulus aggregates. Now, as a rule, learning to react, or *not* to react, to a stimulus combination as distinguished from its components is more difficult than the simple conditioning of a reaction to a stimulus compound. This learning to respond to stimulus combinations or configurations, as such, we shall call the *patterning* of the stimulus compound in question.

For purposes of convenience we shall distinguish two main forms of stimulus patterning. The one to be considered first, because conceptually the simpler, is that in which the onset of the several stimulus energies involved takes place at the same time; this is called *simultaneous stimulus patterning*. The second form is that in which the onset of the several stimulus energies occurs successively; this is called *temporal stimulus patterning*. In some cases the response will be reinforced when it follows the presentation of the stimulus combination; this results in *positive patterning*. In other cases, reinforcement will occur only when response follows the separate presentation of the stimulus components; this results in an extinction of the tendency for the compound to evoke the reaction and is therefore called *negative patterning*.

SOME EXPERIMENTAL EXAMPLES OF SIMULTANEOUS STIMULUS PATTERNING

The first experimental attack on the problems of the patterning of stimulus compounds began in the laboratory of Pavlov, though he did not use this expression in connection with the experiments. Unfortunately he gives no example of simultaneous patterning. He does, however, make this summarizing statement:

It was noticed that if a conditioned reflex to a compound stimulus was established . . . , it was easy to maintain it in full strength and at the same time to convert its individual components, which gave a positive effect when tested singly, into negative or inhibitory stimuli. This result is obtained by constant reinforcement of the compound stimulus, while its components, on the frequent occasions when they are applied singly, remain without reinforcement. This experiment can be made with

ceptor organ. A stimulus aggregate may be defined as a group of stimulus elements all of which usually begin and terminate at the same time, e.g., the stimulus energies given off by an object such as an apple.

equal success in the reverse direction, making the stimulatory compound into a negative or inhibitory stimulus, while its components applied singly maintain their positive effect. (8, p. 144.)

Fortunately there have recently become available some detailed examples of closely analogous forms of simultaneous patterning in dogs; these are from a study reported by Woodbury (10). In one experiment a dog named "Dick" was placed in a wooden stock much like that employed by Pavlov. Just in front of the dog's head was a light horizontal wooden bar; when this bar was raised about half an inch it closed an electrical circuit, activating an electromagnetic device which released into a pan before the dog a pellet of appetizing food. The animal was first taught to lift the bar with his nose to secure the food. Later a wooden shutter was placed before the bar to prevent the dog from nosing it except immediately after the stimuli to be patterned were given. These stimuli were produced by two buzzers, one with a low-pitched, rather raucous tone (represented by *L*), and the other with a definitely higher-pitched and much less raucous tone (represented by

TABLE 11

QUANTITATIVE STATEMENT OF THE COURSE OF THE PATTERNING OF A SIMULTANEOUS STIMULUS COMPOUND LEARNED BY WOODBURY'S DOG, "DICK," TOGETHER WITH THE STEPS IN THE DERIVATION OF THE PATTERNING COEFFICIENT. (Derived from an unpublished table from Woodbury's data, 10.)

Successive Hundreds of Differential Reinforce- ments	Per Cent Reaction Evocation by 50 Presen- tations of Compound <i>HL</i> (<i>Q</i>)	Per Cent Reaction Evocation by 25 Presen- tations of Component <i>H</i>	Per Cent Reaction Evocation by 25 Presen- tations of Component <i>L</i>	Mean Per Cent Reaction Evocation by <i>H</i> and <i>L</i> (\bar{Q})	Patterning Coefficient, $Q - \bar{Q}$ (<i>P</i>)
1	91	91	92	91.5	-.5
2	100	100	100	100.0	0.0
3	100	100	100	100.0	0.0
4	100	100	100	100.0	0.0
5	100	76	100	88.0	12.0
6	100	8	92	50.0	50.0
7	100	12	48	30.0	70.0
8	100	0	40	20.0	80.0
9	100	0	40	20.0	80.0
10	100	0	25	12.5	87.5
11	100	0	0	.0	100.0
12	100	0	4	2.0	98.0
13	100	0	0	.0	100.0

H). The apparatus was so set that when the buzzers were sounded simultaneously (*HL*) and the dog nosed the bar, the food would be delivered, but when either buzzer was sounded alone (*H* or *L*) and the dog nosed the bar, no food would be delivered.

According to Pavlov's summary statement quoted above, this *differential reinforcement*, as it is called, should differentiate the compound from the components. Table 11 and Figure 79 show that such a differentiation did in fact occur and that positive patterning resulted. The dog learned practically never to nose the bar when either *H* or *L* was presented alone but practically always

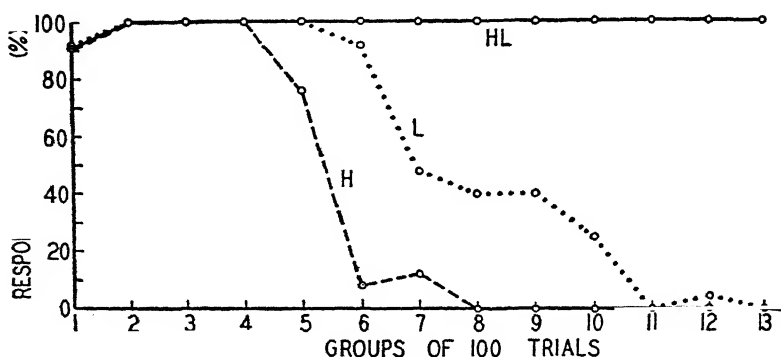


FIG. 79. Graph showing in detail the course of the learning of Woodbury's dog, "Dick," to react positively to a simultaneous stimulus compound consisting of a high-pitched buzzer (*H*) and a low-pitched buzzer (*L*), and not to react to the components, i.e., the buzzers presented separately. Of each one hundred trials, 50 were of the compound (*HL*), 25 were of the high-pitched component, and 25 were of the low-pitched component. This figure was plotted from the values shown in Table 11. (Adapted from Woodbury, 10.)

to nose it at the presentation of the compound, *HL*; this process, however, was a protracted one requiring some 1300 trials. An examination of Figure 79 reveals, in addition to the fact of final differentiation or successful patterning, the following details of the learning process:

1. Although before the introduction of differential training the animal was responding 100 per cent of the time to the stimulus of the bar following the lifting of the shutter, soon after this training began both the compound and the components showed some tendency to extinction or generalized extinction effects, as indicated by the initial depression of all these curves.
2. As practice continued, all three curves rose to 100 per cent, where they remained for some 300 trials.

3. Following this recovery, each component stimulus gradually lost its power to evoke the reaction, the high-pitched buzzer distinctly less slowly than the other.

4. The general shape of the falling curves approximates roughly that of the ogive; i.e., at first the fall is positively accelerated, after which the acceleration reverses itself and becomes negative, finally approaching the horizontal at a zero frequency of response.

In full verification of Pavlov's assertion quoted above, Woodbury reports the detailed record of the pattern learning of a second dog, "Chuck," in which the component stimulus elements in the experimental arrangement just described were positive and the com-

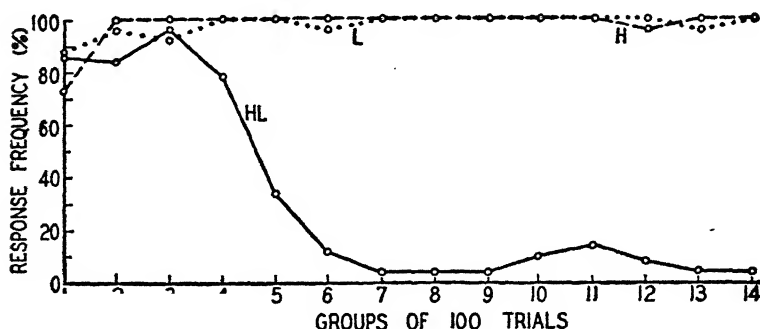


FIG. 80. Graph showing in detail the course of the learning of Woodbury's dog, "Chuck," to react positively to the components of a simultaneous auditory stimulus compound (*H*) and (*L*) and to react negatively to the compound itself (*HL*). Out of each 100 presentations, 50 were *HL*, 25 were *H*, and 25 were *L*.

pound was negative. The course of this bit of negative pattern learning is shown in some detail by the curves of Figure 80. A comparison of this figure with Figure 79 reveals the following:

1. After the first shock of the non-reinforcement associated with the differential training, the positive components rose to 100 per cent and maintained that position substantially to the end of the training, much as did the positive compound in Figure 79.

2. Upon the whole, the components (positive) showed more of a tendency to fall below 100 per cent in Figure 80 than did the compound (positive) in Figure 79.

3. While the negative compound of Figure 80 began to lose its effective excitatory potential more promptly than the negative components in Figure 79, the former showed a greater resistance to complete extinction than the latter.

PRINCIPLES UPON WHICH THE PATTERNING OF STIMULUS COMPOUNDS IS BASED

While Pavlov was completely familiar with the various aspects of the experimental phenomena of patterning, it would appear that he regarded patterning as an ultimate molar phenomenon in that he did not succeed in breaking it down into more elementary principles. This is a little surprising, since he was well acquainted with most, if not all, of the principles required for doing so, notably the principle of afferent neural interaction (see Chapter III, p. 42). Because of the importance of patterning for behavior dynamics, such a derivation will presently be given.

The reader is now familiar with the molar principles upon which the theoretical derivation of the patterning of stimulus compounds is based. For convenient reference they are summarized as follows:

a. Habits are connections between receptor discharges and effector discharges, as shown by the following diagram (p. 111),

$$S - \rightarrow s \text{ --- } r - \rightarrow R,$$

in which the arrow with the broken shaft between s and r represents the habit, or H of the symbol sH_R (Postulate 4, p. 178 ff.).

b. Afferent impulses (s) interact, changing all impulses involved to a greater or less degree, while on their way to that point in the nervous system at which the reinforcement process makes the junction between the s and the r ; the expression \check{s} represents the s as changed by interaction with another s (Postulate 2, p. 47 ff.).

c. When s has become \check{s} , this represents a change in position on a generalization continuum (p. 188). Thus \check{s} would tend to evoke a reaction conditioned to s , though because of the generalization gradient the habit strength, $\check{s}H_R$, and so the reaction potential $\check{s}E_R$, would be weaker than sE_R (Postulate 5).

d. In the case of complete experimental extinction, the total inhibition is equal to the difference between the reaction potential and the reaction threshold, sL_R (p. 323 ff.), i.e.,

$$I_R = sE_R - sL_R.$$

e. The gradients of generalization of both excitatory potential, sE_R , and inhibitory potential, sI_R , have approximately the same slope; i.e., their equations have the same exponent (p. 275 ff.).

f. Other things equal, the magnitude of the interaction effects between afferent impulses from a single receptor field, e.g., the retina, will be greater than that between impulses which arise from distinct receptor fields, e.g., the retina and the tactile receptors.

g. Other things equal, the magnitude of the interaction effect of one afferent impulse upon a second is an increasing monotonic function of the magnitude of the first (Postulate 2, p. 47 ff.).

h. When a stimulus energy ceases to act on a receptor, the conditionable afferent impulses set in motion by the stimulation continue to be active in the central neural substance for some seconds; the intensity of this perseverative tendency gradually diminishes as a negative growth function of the time since the termination of the action of the stimulus on the receptor (Postulate 1, p. 47).

i. The empirical degree of stimulus patterning (P) where the reaction is of the all-or-none type (such as that displayed by Woodbury's dogs) may be represented sufficiently well for our present expository purposes by the formula,

$$P = Q - \bar{Q}, \quad (52)$$

where Q represents the mean empirical per cent of reaction evocations by the reinforced phase of the compound, and \bar{Q} represents the mean per cent of reaction evocations by the negative or non-reinforced phase of the compound.

j. The theoretical degree of stimulus patterning (P') may be conveniently represented for our present purposes by the formula,

$$P' = 100 \left(1 - \frac{\bar{Q}'}{Q'} \right), \quad (53)$$

in which \bar{Q}' is the effective reaction potential of the negative portion of the discrimination, and Q' is the effective reaction potential of the positive portion of the discrimination.

Both the P and the P' formulas yield a value of 100 for perfect or complete patterning, and one of zero where no patterning tendency exists. In case patterning is negative, both formulas yield a negative value. Several examples of the use of the second, or P' formula, will be encountered in the immediately following pages. An extended example of the use of the empirical or P formula is contained in Table 11. Substituting appropriately in this formula from the first row of entries, we have,

$$\begin{aligned} P &= 91 - 91.5 \\ &= - .5, \end{aligned}$$

which yields a slight and presumably atypical negative value; this value constitutes the first entry in the last column. The patterning index or coefficient, P , reveals effectively the course of the acquisition of patterning as a unified process. This is brought out strikingly by the graphic representation of the P -values as a

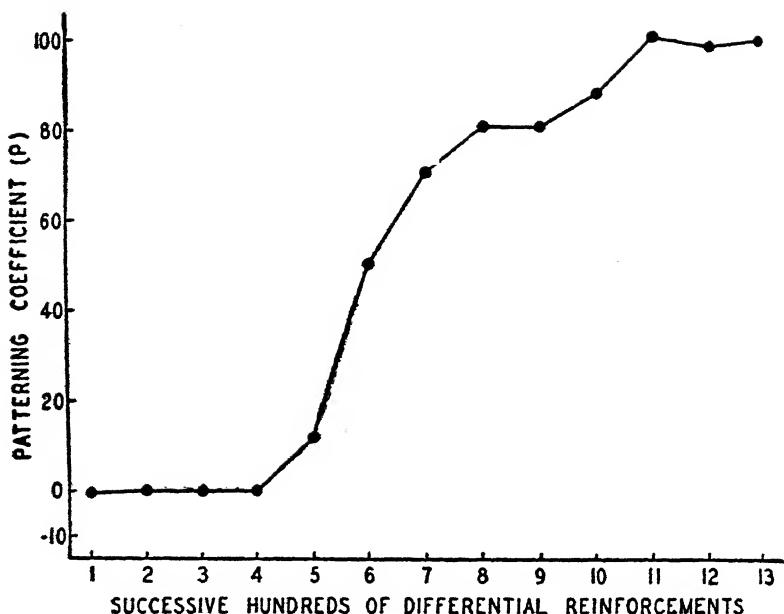


FIG. 81. Graphic representation of the progress of the positive patterning of a simultaneous stimulus compound learned by Woodbury's dog, "Dick." Plotted from the final column of Table 11. Note the approximately ogival form of this curve. (See Chapter XIII, p. 204 ff.)

function of the number of differential reinforcements, which may be seen in Figure 81.

THEORETICAL DERIVATION OF THE SPONTANEOUS OR QUASI-PATTERNING OF SIMULTANEOUS STIMULUS COMPOUNDS

Proceeding to the application of the above principles to specific values, we shall first make a theoretical analysis of the tendency to spontaneous or *differentially* unlearned patterning exhibited by a simple conditioned simultaneous stimulus compound in which the components are potentially negative, i.e., *about to be* unreinforced. In this way will be rounded out a discussion begun above (Chapter XIII, p. 204) concerning the influence of afferent interaction on the dynamics of stimulus compounds (p. 217 ff.).

Let it be assumed that by the Pavlovian technique a simultaneous stimulus compound of two components (S_1 and S_2) is conditioned to a reaction (R) strongly enough for S_1 to command a

habit strength of 40 habs and S_2 to command a habit strength of 60 habs. Taking the value of the effective drive, \bar{D} (Chapter XIV, p. 245), at unity we have,

$$\check{s}_1 E_R = 40 \text{ wats}$$

and

$$\check{s}_2 E_R = 60 \text{ wats.}$$

Now, by the physiological summation of these two excitatory potentials, we have,

$$\begin{aligned} Q' &= \check{s}_1 + \check{s}_2 E_R = 40 + 60 - \frac{40 \times 60}{100} \\ &= 100 - 24 \\ \therefore Q' &= 76 \text{ wats.} \end{aligned}$$

We next consider the excitatory potential of the two components if each is acting alone, i.e., independently. Since the respective afferent impulses will not be interacting upon the separate presentations of S_1 and S_2 , the afferent impulses resulting from their individual action on the receptors will be represented simply by s_1 and s_2 , i.e., without the breve. Now (Postulate 5), the shift from the \check{s}_1 of the compound to the s_1 of separate action will involve a generalization effect and therefore a fall in excitatory potential from $\check{s}_1 E_R$ to $s_1 E_R$. Let it be assumed that throughout this particular problem, unless otherwise stated, the reduction from \check{s} to s , or vice versa, amounts to 25 per cent. Accordingly, we have as the separate action of the respective components,

$$s_1 \bar{E}_R = 40 - 10 = 30 \text{ wats}$$

$$s_2 \bar{E}_R = 60 - 15 = 45 \text{ wats;}$$

and, by calculated physiological summation, these two potentially negative stimulus components would exert the equivalent of,

$$\begin{aligned} s_1 + s_2 \bar{E}_R &= 30 + 45 - \frac{30 \times 45}{100} \\ &= 75 - 13.5 \\ \therefore \bar{Q}' &= 61.5 \text{ wats.} \end{aligned}$$

Next, substituting in the formula for calculating the theoretical patterning index (P'), we have,

$$\begin{aligned} P' &= 100 \left(1 - \frac{61.5}{76.0} \right) \\ &= 100 (1 - .809) \\ &= 100 \times .191 \\ &= 19.1. \end{aligned}$$

This means that *at the very outset of the patterning process, in case the components are negative, afferent interaction produces a small amount of spontaneous positive patterning.*

We turn next to the theoretical analysis of the spontaneous patterning of a simultaneous stimulus compound in which the components are separately reinforced and the compound is potentially negative. In that event, making assumptions analogous to those of the preceding case, we have,

$${}_1E_R = 40 \text{ wats,}$$

and

$${}_2E_R = 60 \text{ wats,}$$

which if summated physiologically would yield,

$$Q' = {}_1 + {}_2E_R = 76.$$

Similarly, by the process of generalization from s to \tilde{s} , each excitatory potential in passing from the separate state to that of the compound would reduce the reaction potentials from 40 to 30 wats, and from 60 to 45 wats respectively, which by physiological summation would yield,

$$\bar{Q}' = \tilde{r}_1 + \tilde{r}_2E_R = 61.5,$$

just as before.

Substituting these Q -values in the formula for patterning, we have,

$$P' = 19.1,$$

exactly as when the compound was reinforced. These considerations lead us to our first corollary:

I. *Following the application of positive reinforcement but previous to the application of unreinforcement, situations involving simultaneous stimulus compounds will show a certain tendency to*

patterning, the strength of the tendency being the same whether the compound or the components are reinforced.

We turn now to the consideration of the influence on the amount of spontaneous patterning of increasing the amount of neural interaction effect. Clearly, the greater the amount of interaction, the greater will be the reduction in generalization from s to \check{s} and vice versa. Let it be supposed that this reduction is increased from the 25 per cent assumed above, to 50 per cent. In that case, on assumptions otherwise the same as above, if the compound is positive we have after generalization,

$$\check{s}_1 \bar{E}_R = 40 - 20 = 20,$$

$$\check{s}_2 \bar{E}_R = 60 - 30 = 30.$$

By physiological summation, assuming the components to be negative, we have,

$$\begin{aligned} \bar{Q} &= 20 + 30 - \frac{20 \times 30}{100} \\ &= 50 - 6 \\ &= 44. \end{aligned}$$

Since, exactly as when the generalization reduction was 25 per cent,

$$Q = \check{s}_1 + \check{s}_2 \bar{E}_R = 76,$$

we have, by substituting in the patterning formula,

$$\begin{aligned} P' &= 100 \left(1 - \frac{44}{76} \right) \\ &= 100 (1 - .578) \\ &= +42.2. \end{aligned}$$

This value of $+42.2$, when the generalization reduction due to neural interaction is 50 per cent, is to be compared with the smaller P -value of 19.1 when the generalization reduction is 25 per cent. Since, by Corollary I, the spontaneous tendency to patterning is the same when the components are positive, it will not be necessary to derive that case for the decreased rate of generalization.

Generalizing from the above calculation, we arrive at our second corollary:

II. *The greater the amount of afferent neural interaction, the greater will be the amount of "spontaneous" patterning, both positive and negative.*

THEORETICAL DERIVATION OF THE POSITIVE PATTERNING OF
SIMULTANEOUS STIMULUS COMPOUNDS BY
DIFFERENTIAL REINFORCEMENT

The theoretical derivation of quasi or spontaneous patterning has much in common with that of the genuine patterning of stimulus compounds which is attained by means of differential reinforcement. Accordingly the above account of the former will serve as a useful introduction to the slightly more complicated derivation of the latter now to be given. The two processes have in common a dependence upon afferent interaction with the consequent operation of the generalization gradient, physiological summation, etc. Genuine patterning involves the additional complication of the generation of experimental extinction (I_R) which necessarily results from the non-reinforcement that is a part of differential reinforcement.¹ There is also involved the associated generation of conditioned inhibition (sI_R) and the generalization of a portion of this back upon the positive or reinforced phase of the patterning situation.

We shall take as our first example the case in which the compound is positive (reinforced). In order to make the exposition as intelligible as possible, we shall assume, as in the above examples, that we have a compound of only two stimulus components, S_1 and S_2 , and that the process begins with the initial conditioning of the compound to the reaction, R ; this yields, as before, excitatory potentials as follows:

$$\gamma_1 E_R = 40 \text{ wats}$$

$$\gamma_2 E_R = 60 \text{ wats,}$$

which, by physiological summation, yields a $\gamma_1 + \gamma_2 E_R$ of 76 wats (p. 223). Now, by generalization, $\gamma_1 E_R$ shrinks 25 per cent from a value of 40 to one of 30 and $\gamma_2 E_R$ shrinks from a value of 60 to one of 45, i.e.,

$$\gamma_1 E_R = 30 \text{ wats}$$

and

$$\gamma_2 E_R = 45 \text{ wats.}$$

Assuming a reaction threshold of 10 wats as a minimum necessary to evoke reaction (see p. 323 ff.), the non-reinforcement of the

¹The generation of inhibition during reinforcement is here neglected in the interest of expository simplicity.

separate components s_1E_R and s_2E_R to the reaction threshold (10 wats) would reduce each to a value of zero *responses*, with the generation of inhibition amounting to,

$$30 - 10 = 20 \text{ pavs}$$

and

$$45 - 10 = 35 \text{ pavs.}$$

Assuming that half of each of these values is made up of conditioned inhibition (sI_R) and therefore is subject to stimulus generalization (p. 281 ff.), and recalling that, by assumption, 75 per cent stimulus generalization occurs between s and \bar{s} , or vice versa, we have as the amount of inhibitory potential generalizing from the respective non-reinforcements back upon the corresponding reinforcement phase,

$$\bar{s}_1\bar{I}_R = \frac{20}{2} \times .75 = 7.5 \text{ pavs}$$

$$\bar{s}_2\bar{I}_R = \frac{35}{2} \times .75 = 13.13 \text{ pavs.}$$

Summating these inhibitory potentials physiologically, we have,

$$\begin{aligned}\bar{s}_1 + \bar{s}_2\bar{I}_R &= 7.5 + 13.13 - \frac{7.5 \times 13.13}{100} \\ &= 20.63 - .99 \\ &= 19.64 \text{ pavs.}\end{aligned}$$

From this it follows from the formula for $s\bar{E}_R$ that,

$$\begin{aligned}\bar{s}_1 + \bar{s}_2\bar{E}_R &= 76 - 19.64 \\ &= 56.36,\end{aligned}$$

i.e.,

$$Q' = 56.36 \text{ wats.}$$

On the other hand, since the two threshold values left by the extinction process never operate together, they will be averaged, rather than summated, i.e.,

$$10 \text{ wats.}$$

Substituting these Q -values in the formula for patterning, we have,

$$\begin{aligned} P' &= 100 \left(1 - \frac{10}{56.36} \right) \\ &= 100 (1 - .177) \\ &= 82.3, \end{aligned}$$

which represents a large degree of patterning.

Suppose, on the other hand, that we employ the empirically more realistic response formula for observed patterning,

$$P = Q - \bar{Q}.$$

The per cent of responses to $s_i E_R$ equals 10 wats (after extinction to zero), and $s_s E_R$ equals the same. But,

$$sL_R = 10,$$

and when

$$sE_R = sL_R,$$

$$p = 0,$$

i.e.,

$$\bar{Q} = 0.$$

Next, if we assume that the maximum range of oscillation (sO_R) is 40, since,

$$Q' = 56 \text{ wats}$$

and the threshold is taken as 10 wats, it follows that oscillation will frequently carry the reaction potential below the reaction threshold (sL_R), i.e.,

$$56 - 10 > 40,$$

from which it follows that the $s_i + s_s \bar{E}_R$ will yield 100 per cent reaction evocations, and the value of Q will be 100 per cent. Substituting these values in the formula for P , we have,

$$\begin{aligned} P &= Q - \bar{Q} \\ &= 100 - 0.00 \\ &= 100; \end{aligned}$$

i.e., empirical patterning (P) will be perfect. Thus we arrive at our third corollary:

III. In case a simultaneous stimulus compound and its components receive differential reinforcement, the components being negative (unreinforced), sufficient training will produce complete empirical patterning provided the differences between \check{s} and s are great enough to bring about a residual $\check{s}_1 + \check{s}_2 E_R$ which exceeds the range of behavioral oscillation (sO_R) plus the reaction threshold (sL_R).

THEORETICAL DERIVATION OF THE NEGATIVE PATTERNING OF SIMULTANEOUS STIMULUS COMPOUNDS BY DIFFERENTIAL REINFORCEMENT

We turn next to the case of the patterning of a simultaneous stimulus compound in which all the conditions are assumed to be the same as those in the preceding example, except that the compound is negative, i.e., S_1 and S_2 are reinforced separately until

$${}_1E_R = 40 \text{ wats}$$

and

$${}_2E_R = 60 \text{ wats.}$$

Then S_1 and S_2 are presented as a compound and given differential reinforcement. By reasoning exactly analogous to that of the preceding example, s will generalize to the \check{s} of the compound to the extent of 75 per cent, yielding, when in the compound at the outset of differential reinforcement,

$$\check{s}_1 E_R = 30 \text{ wats}$$

and

$$\check{s}_2 E_R = 45 \text{ wats.}$$

Since these are extinguished jointly in $\check{s}_1 + \check{s}_2 E_R$, each will contribute roughly equal amounts to the 10-wat threshold, i.e., $\bar{Q}' = 10$. This will leave to be extinguished by the negative aspect of differential reinforcement, and so to be converted into inhibition,

$$30 - 5 = 25 \text{ pavs}$$

and

$$45 - 5 = 40 \text{ pavs.}$$

If, now, half of this inhibition in each case is sI_R and 75 per cent of sI_R generalizes, we have,

$$s_1\bar{I}_R = \frac{25}{2} \times .75 = 9.38 \text{ pavs,}$$

$$s_2\bar{I}_R = \frac{40}{2} \times .75 = 15.00 \text{ pavs}$$

to generalize back on s_1E_R and s_2E_R respectively. It follows that,

$$s_1\bar{E}_R = 40 - 9.38 = 30.62 \text{ wats}$$

and

$$s_2\bar{E}_R = 60 - 15 = 45.00 \text{ wats.}$$

Since the two negative potentials are generated together, they are already summated physiologically at the reaction threshold, i.e.,

$$\bar{Q} = 10 \text{ wats.}$$

On the other hand, since s_1E_R and s_2E_R never operate together, they will be averaged:

$$Q' = \frac{30.62 + 45.00}{2}$$

$$\therefore Q' = 37.81 \text{ wats.}$$

Substituting these Q -values in the patterning formula, we have,

$$\begin{aligned} P' &= 100 \left(1 - \frac{10}{37.81} \right) \\ &= 100 (1 - .264) \\ &= 100 (.736) \\ &= 73.6, \end{aligned}$$

which represents a considerable patterning tendency.

Suppose, now, we consider the response aspects of this situation. It may be seen that, assuming the range of behavioral oscillation (sO_R) to be 40 wats, since, as stated above,

$$s\bar{E}_R = 45 \text{ wats,}$$

and since

$$sL_R = 10 \text{ wats,}$$

and

$$45 - 10 < 40,$$

S_2 will not evoke R 100 per cent of its presentations but, by Table 9, would yield about 96 per cent of reaction evocations.¹ Also since

$$s_1 \bar{E}_R = 30.62 \text{ wats,}$$

$$sL_R = 10 \text{ wats,}$$

and since

$$32.62 - 10 < 40,$$

it follows that S_1 will evoke R at less than 100 per cent of its presentations, i.e., at approximately 61 per cent of the stimulations. Thus,

$$Q = \frac{96 + 61}{2} = 78.5.$$

Accordingly, substituting in the empirical patterning formula we have,

$$\begin{aligned} P &= 78.5 - 0.00 \\ &= 78.5, \end{aligned}$$

a degree of response patterning appreciably less than perfect and so definitely less than the degree of response patterning found under comparable conditions where the compound was positive. It is evident, however, that if $s_1 H_R$ and $s_2 H_R$ were sufficiently reinforced to bring each of them up to a value of about 70 habs, both $s_1 \bar{E}_R$ and $s_2 \bar{E}_R$ would mediate the evocation of R on 100 per cent of the trials, in which case the empirical patterning index would be 100, i.e., perfect even under the conditions of component reinforcement. This brings us to the statement of our fourth corollary:

IV. *In case a simultaneous stimulus compound and its components receive differential reinforcement, the compound being unreinforced, sufficient training will produce complete empirical patterning provided the differences between s and \bar{s} are great enough to produce a residual $s \bar{E}_R$ which exceeds the range of behavioral oscillation (sO_R); but, other things equal, this type of learning will require a greater number of differential reinforcements to attain a given degree of patterning than will that in which the components are unreinforced.*

¹ The statement of the method of determining this value and the reasons for so doing were indicated above, p. 328 ff. It may be said here, however, that the major assumption underlying the procedure is that the value of $s \bar{E}_R$ oscillates below its expressed potential magnitude and that the calculation involved the use of the probability function of behavioral oscillation shown in Table 9 (p. 311).

The tendency of empirical patterning to break down through the failure of the components to evoke the reaction at 100 per cent of the stimulations is nicely illustrated in Figure 80, where, at the twelfth hundred differential reinforcements, we have,

$$\bar{Q} = \frac{96 + 100}{2} = 98 \text{ per cent.}$$

At the same time,

$$Q = 8 \text{ per cent,}$$

which yields as an index of patterning,

$$P = 98 - 8 = 90,$$

a value distinctly less than perfection.

THE POSITIVE PATTERNING OF SIMULTANEOUS STIMULUS COMPOUNDS UNDER CONDITIONS OF DECREASED GENERALIZATION

We now come to the final step in our analysis of the process of stimulus-patterning learning by differential reinforcement. Let us consider the influence on this process of an increase in the afferent interaction effects sufficient to steepen the *fall* in the generalization gradient from 25 per cent to 50 per cent. In order to simplify the exposition we shall take the original case of a simultaneous stimulus compound in which the compound is first reinforced so that,

$${}_1E_R = 40 \text{ wats}$$

and

$${}_2E_R = 60 \text{ wats.}$$

Now, by generalization, with a loss of 50 per cent, it follows that,

$${}_1E_R = 20 \text{ wats}$$

and

$${}_2E_R = 30 \text{ wats.}$$

The separate extinction of each of these to the reaction threshold of 10 wats would generate inhibition in the amount of

$$20 - 10 = 10 \text{ pavs}$$

and

$$30 - 10 = 20 \text{ pavs.}$$

Assuming that half of each of these generalizes to the extent of 50 per cent, we have,

$$\check{s}_1 \bar{I}_R = \frac{10}{2} \times .50 = 2.5 \text{ pavs}$$

and

$$\check{s}_2 \bar{I}_R = \frac{20}{2} \times .50 = 5.0 \text{ pavs.}$$

Summating these inhibition values,

$$\begin{aligned}\check{s}_1 + \check{s}_2 \bar{I}_R &= 2.5 + 5.0 - \frac{2.5 \times 5.0}{100} \\ &= 7.5 - .01 \\ &= 7.49 \text{ pavs.}\end{aligned}$$

It follows that

$$\begin{aligned}\check{s}_1 + \check{s}_2 \bar{E}_R &= 76 - 7.49 \\ &= 68.51,\end{aligned}$$

i.e.,

$$Q' = 68.51 \text{ wats.}$$

As in the example involving 25 per cent generalization, the threshold reaction potentials remaining in $s_1 E_R$ and $s_2 E_R$ are 10 wats each.

$$\bar{Q}' = \frac{10 + 10}{2} = 10 \text{ wats.}$$

Substituting in the following formula, we have,

$$\begin{aligned}P' &= 100 \left(1 - \frac{10}{68.51} \right) \\ &= 100 (1 - .146) \\ &= 100 \times .854 \\ &= 85.4.\end{aligned}$$

This value, it will be noted, is larger than the 82.3 yielded by the smaller fall in the generalization gradient.

However, just as in the case of the 25 per cent generalization reduction, both components, despite the threshold values of 10 pavs, will yield 0.00 per cent of reaction evocation, i.e.,

$$\bar{Q} = \frac{0.00 + 0.00}{2} = 0.00.$$

On the other hand, since

$$68.51 - 10 > 40,$$

the compound will evoke R on 100 per cent of the presentations, i.e.,

$$Q = 100.$$

Accordingly,

$$\begin{aligned} P &= 100 - 0.00 \\ &= 100; \end{aligned}$$

i.e., empirical patterning will be perfect. This brings us to our fifth corollary:

V. In the patterning of stimulus compounds, the greater the fall in the generalization gradient between \bar{s} and s , the less will be the difficulty in attaining a given degree of discrimination.

SOME EXPERIMENTAL EXAMPLES OF THE LEARNING OF TEMPORAL STIMULUS PATTERNS

We turn now to the problem of the patterning of temporal sequences of stimuli, i.e., the learning of temporal stimulus patterns. Pavlov, in whose laboratory this type of learning seems first to have been studied, describes several such experiments. In an experiment performed by Dr. Eurman, a dog was presented with a sequence of three stimuli: a light (L), a cutaneous stimulus (C), and the sound of bubbling water (S). When these stimuli were given in the order LCS , the presentation was always followed by food reinforcement, but when they were given in the reverse order, i.e., SCL , the combination was never reinforced. The dog finally reached a point of training such that it secreted an average of about 8 drops of saliva to LCS , and zero drops to SCL , which presumably indicated perfect patterning. Unfortunately, Pavlov does not report the course of the learning of this or any other pattern, though he makes a general statement that such learning often requires protracted training and when achieved is very unstable (8, p. 147).

Fortunately we have in the study by Woodbury (10) already referred to, a detailed report of the course of both the positive and the negative forms of temporal patterning. The same apparatus arrangement and general procedure were employed as described above for the patterning of simultaneous conditioned com-

pounds, but with this difference: the high-pitched buzzer was sounded for one second; then, after a pause of one second, the low-pitched buzzer was sounded for one second, following which the shutter was raised, giving the dog access to the bar. If the dog nosed the bar, the act was reinforced by food. On the negative side, the high-pitched buzzer was presented for one second twice in succession, with a pause of one second between presentations. On other occasions the low-pitched buzzer would be presented twice in the same way. After each of these presentations the shutter was lifted, but if the dog nosed the bar no food would be given. The learning behavior of the dog "Ted," by this procedure, is shown in detail by Figure 82. A study of this figure indicates in general

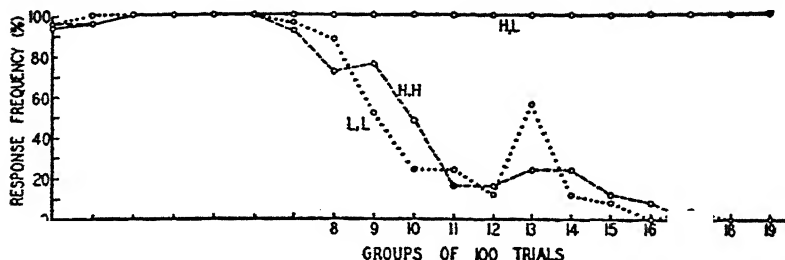


FIG. 82. Graph showing in detail the course of the learning of Woodbury's dog, "Ted," to react positively to a temporal stimulus compound of a high-pitched buzzer followed by a low-pitched buzzer (H,L), and negatively to a parallel presentation of the high-pitched buzzer (H,H), and of the low-pitched buzzer (L,L). Out of each 100 presentations, 50 were H,L , 25 were H,H , and 25 were L,L . (Adapted from Woodbury, 10.)

a striking agreement with the course of the patterning of simultaneous stimulus compounds, except that the amount of training required to complete the process of temporal patterning was noticeably greater. There is the same initial depression of all three curves at the outset of differential reinforcement, and the same subsequent rise of all three to 100 per cent; following this the two non-reinforced component combinations gradually fall toward zero, taking a roughly ogival course.

The exact reverse of the experiment just described was carried out by Woodbury with the dog "Bengt"; i.e., the temporal sequences H,H and L,L were reinforced, but the sequence H,L was never reinforced. The course of this learning may be seen in Figure 83, a study of which reveals the same general features as those of Figure 80, though the difficulty of learning is somewhat

greater. It may also be noted that Figures 80 and 83 both show a greater amount of disturbance (weakening) of the reinforced phases than do Figures 79 and 82; this increased disturbance presumably comes from the generalization of the greater amount of inhibition arising from the extinction of the non-reinforced compound upon the relatively weak component.

It is to be noted that even though the values represented in the preceding figures are derived from the pooling of a very large number of observations, the performance of a single animal is not sufficient basis for the establishment of empirical laws, though it

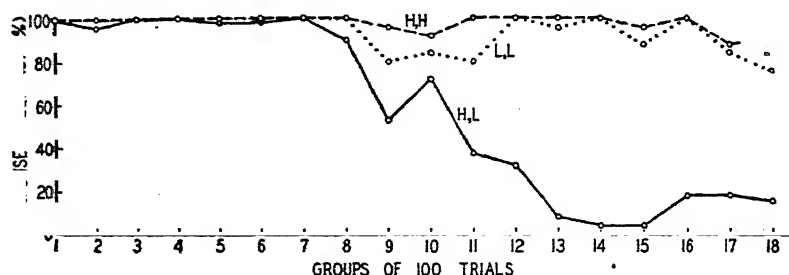


FIG. 83. Graph showing in detail the course of learning of Woodbury's dog, "Bengt," to react positively to the temporal combinations *H,H* and *L,L*, and to react negatively to the combination *H,L*. Out of each 100 presentations, 100 were *H,L*, 25 were *H,H*, and 25 were *L,L*. (Adapted from Woodbury, 10.)

may serve to illustrate theoretical principles; it is as such that Woodbury's graphs are offered here for consideration. However, the performances of these single animals give complete and sufficient proof of one thing: they demonstrate that the positive and the negative forms of both types of stimulus patterning *can* be learned by dogs.

A THEORETICAL ANALYSIS OF TEMPORAL STIMULUS PATTERNING

The theoretical analysis of temporal stimulus patterning is at bottom about the same as that of simultaneous stimulus patterning. This is to say that temporal stimulus patterning depends upon substantially the same principles: afferent neural interaction, the generalization of excitation, the extinction of the generalized excitation, and the final generalization of this inhibition back upon the positive reaction potential. There is, however, this important difference: in temporal stimulus patterning the neural interaction

presumably takes place between the afferent impulses arising directly from the second stimulation and the *perseverative stimulus traces* (Postulate 1) which were originally set in motion by the earlier stimulation. Thus the neural interaction of the components of temporal stimulus patterns is a simultaneous affair exactly as is that of the components of simultaneous patterning; the difference lies in the fact of the temporal asynchronism of the action of the respective stimulus energies which originally set the interacting impulses in motion. From the point of view of adaptation and survival this difference is of enormous importance, but the patterning mechanism itself differs little except quantitatively.

It is evident from the foregoing that having postulated perseverative stimulus traces (Postulate 1), the patterning of temporal stimulus compounds follows at once, by reasoning exactly analogous to that by which Corollary III was derived. This brings us to the statement of our sixth corollary:

VI. *In case (1) a temporal stimulus compound and (2) a repetition of either component in the same tempo as that of the presentations of the compound, receive differential reinforcement, the repetition of the component being unreinforced, sufficient training will produce complete stimulus patterning provided the difference between s and \check{s} is great enough to bring about a residual $s_1 + s_2 \bar{E}_R$ which exceeds the reaction threshold by an amount greater than the range of behavioral oscillation (sO_R).*

Having derived the basic phenomenon of temporal stimulus patterning, we proceed to the examination of certain quantitative differences which may, theoretically, be expected to manifest themselves in the comparison of the patterning of simultaneous and successive stimulus compounds. The most striking of these differences is evidently due to the progressive diminution in the intensity of the stimulus trace with the passage of time, as stated in Principle *h* (Postulate 1); i.e., other things equal, the intensity of a stimulus trace will be weaker than the original afferent impulse set in motion by the action of the stimulus energy (S) on the receptor. Also, by Principle *g* (Postulate 1), a weak afferent impulse (s_1) will produce a smaller interaction effect on a second afferent impulse (s_2) than will a strong value of s_1 . But the smaller the difference between s and \check{s} , the less will be the fall in the generalization gradient; and, by Corollary V, the smaller the fall in the generalization gradient between s and \check{s} , the greater will be the difficulty in the attainment of a given degree of pattern dis-

crimination. From these considerations there follow our seventh and eighth corollaries:

VII. *Other things equal, a given degree of the patterning of temporal stimulus compounds will require more differential reinforcements than will that of simultaneous stimulus compounds.*

VIII. *Other things equal, in the patterning of a temporal stimulus compound, the greater the lapse of time between the termination of one stimulus and the beginning of the next, the greater will be the number of differential reinforcements required to attain a given degree of patterning.*

THE RESOLUTION OF HUMPHREY'S ARPEGGIO PARADOX

A good deal of misunderstanding has arisen in learning theory regarding the rôle performed by the stimulus, presumably because of the hidden nature of neural interaction effects. This may be illustrated by an experiment reported by Humphrey (5, pp. 198, 237):

Subjects were trained . . . to raise their hand at the sound of a certain specific tone. The training was accomplished by administering an electric shock when the tone was sounded, but never when any other tone was sounded. Suppose that the active tone was G above middle C. We have then a conditioned reflex to this tone, which has been differentiated out so that no other tone producible on the apparatus was followed by response. By prolonged practice this conditioned reflex became very highly stabilized. Suppose now that the active note is included in a melody or an arpeggio such as that formed by the successive notes C, E, G, C, where G is the active note. [The instrument was of the xylophone type with metal cylinders, which were not damped after a note was struck, consequently the vibrations from one note persisted during the striking of the next note, as in legato piano playing. (p. 198.)] The arpeggio then contains the stimulus for the conditioned response. The records show that while the isolated note was consistently followed by response, the same note, repeated immediately in an arpeggio, was consistently not followed by response. The melody "Home Sweet Home" when played in the key of C contains the note G fourteen times. Experiments showed that subjects trained to this note consistently respond to it when presented in isolation and do not respond to it when presented in the melody. This result is very striking in view of the fact of the fourteen repetitions of the active note included in the melody as played. (p. 237.)

How shall these experimental observations be interpreted? The above account seems to show that when the note G was struck, the preceding notes C and E were also still vibrating, which would

produce a simultaneous stimulus compound. In the case of the melody, "Home Sweet Home," presumably sometimes there would be a simultaneous stimulus compound of the "active" note and the two or three preceding notes combined with a temporal compound made up of the impulses from these active stimuli and the *perseverative stimulus traces* arising from the stimulations of the more remotely preceding notes. Since, according to the preceding analysis, afferent impulses and perseverative traces operate in much the same way, an analysis of the simpler arpeggio situation will suffice for both.

According to the neural interaction hypothesis, the afferent impulse, s_C , would be changed to the impulse \check{s}_C when conjoined with the impulses arising from S_C and S_E . This alone might easily weaken the reaction potential $s_G E_R$ sufficiently to produce external inhibition. However, it must be recalled that, by differential reinforcement, there had presumably been developed a considerable amount of conditioned inhibition, $s_C I_R$ and $s_E I_R$. Let it be supposed, for example, that $s_G E_R$ has a strength of 50 wats and that $s_C I_R$ and $s_E I_R$ have strengths of 30 pavs each. Because of the progressive damping by the air, the intensity of S_C and of S_E will be reduced; this should be especially true of S_C , since it was struck first. Therefore, both $s_C I_R$ and $s_E I_R$ will be weakened appreciably, $s_C I_R$ to 20 pavs, say, and $s_E I_R$ to 25 pavs. Now, as the result of afferent interaction and the consequent fall in the generalization gradient, both the excitatory and the inhibitory tendencies alike will suffer a certain reduction in strength when they enter the compound, say 20 per cent. This leaves us with the following values:

$$\check{s}_G E_R = 40 \text{ wats}$$

$$\check{s}_C I_R = 16 \text{ pavs}$$

$$\check{s}_E I_R = 20 \text{ pavs.}$$

Summating the two inhibitions, we have,

$$\begin{aligned} \check{s}_C + \check{s}_E I_R &= 16 + 20 - \frac{16 \times 20}{100} \\ &= 36 - 3.2 \\ &= 32.8. \end{aligned}$$

It therefore follows that the effective reaction potentiality to the lifting of the hand at the striking of the note G in the midst of the arpeggio will be,

$$s_G \bar{E}_R = 40 - 32.8 \\ = 7.2.$$

If we assume, as in the preceding computations (p. 364), that the reaction threshold has a value of 10 wats, it appears that under the given conditions the net effective reaction potential available for reaction evocation in the arpeggio (7.2 wats) would be below the reaction threshold and therefore the hand would not be lifted during the playing of the arpeggio, exactly as Humphrey found. Thus Humphrey's auditory configurational problem finds a natural and consistent explanation in terms of habit dynamics.¹

SOME GENERAL CONSIDERATIONS CONCERNING THE FUNCTIONAL DYNAMICS OF STIMULUS PATTERNS

From the point of view of causation, an organism and its entire environment must be regarded as a complex causal interacting unit (1). Probably in all adaptive situations the act of the organism does not yield an effect which produces a particular type and amount of reinforcement until every one of a number of different conditions is satisfied. In an ideal adaptive situation the organism would have (a) receptors which would respond differentially to the impact of energies characteristic of each of the several critical conditions, and (b) receptors responsive to each of the various critical conditions which would *prevent* the act from resulting in reinforcement; also (c) the stimulus energies associated with these several conditions should actually impinge on the relevant receptor, e.g., they should not be shielded from the receptor by the interposition of some other object. Under these ideal conditions, patterning would seem to be the only effective form of habit structure. Unfortunately, even though for the most part conditions a and b obtain with higher organisms, condition c is often satisfied not only imperfectly but to varying and fortuitous degrees of imperfection.

¹ It may be added that the context of the quotation from Humphrey cited above seems to indicate that no small portion of the confusion in this case comes from the purely semantic difficulty of having failed to recognize, and symbolize, the distinction between the stimulus energy (S) and the afferent impulse (s).

For this reason the question of whether a reaction in a given situation will be followed by reinforcement is almost always more or less of a gamble for the organism, even in the most advanced stages of training. It thus comes about, as Brunswik (1) has pointed out, that as the number of critical stimulus cues increase, the probability becomes greater that the total group or configuration of causal factors necessary for the act to eventuate in reinforcement is present in fact. Therefore, as noted in connection with the habit dynamics of stimulus compounds, the summation of habit strengths which are based on the afferent impulses of the stimulus components, s , as contrasted with the interaction effects represented by \hat{s} , is a *primitive biological first-approximation to a calculus of adaptive probability*. This mechanism, coupled with that of the reaction threshold (sL_R), prevents the organism from wasting its energy by reacting when the probability of need reduction is too slight. Similarly, if the number of times that a given stimulus element or aggregate has been associated with reinforcement is small, the habit strength will be small, the probability of response evocation will be small, and so here again the organism will tend to react automatically to the probabilities of the situation. The implications of the very numerous permutations of these and related factors cannot be entered into here, though many of them are fairly obvious.

But in case a situation is sufficiently stabilized for the critical variable factors to satisfy condition c , as was the case with Woodbury's dogs, experiments show, on the basis of recognized principles, that the organism can largely transcend the initial crude summation calculus of adaptive probabilities by reacting, or not, with precision to particular combinations or configurations of stimulus aggregates. It is true that a reaction associated with a particular pattern discrimination developed in one static setting may not be followed by reinforcement in another, so that the organism must continue to gamble to some extent as long as life lasts. However, each new reaction brings with it increased training in discrimination, which makes the odds fall progressively more in the animal's favor as life goes on.

One favoring subsidiary factor is that by compound trial and error organisms learn to adjust their receptors in such a way as to expose them more adequately to all the environmental energies relevant to a given need; this is the behavioral mechanism which brings about searching or exploration. Another favoring behavioral

mechanism, at least in human organisms, is the conditioning by social trial and error of characteristic symbolic acts such as words, to certain stable and significant stimulus aggregates (objects); this presumably facilitates very greatly the indirect generalization (p. 191) of instrumentally adaptive reactions set up in one configurational situation which enables them to function in other situations adaptively similar but differing to a considerable degree in stimulus configurational characteristics. Probably this explains why Razran (9) found with verbally sophisticated human subjects that configurational generalization was considerably wider than was simple stimulus generalization. Feeble-minded individuals and dogs might be expected to show rather different results.

On the basis of the above considerations the conjecture is hazarded that one of the more important capacities in the higher levels of intelligence is that of discriminating afferent interaction effects. It is believed that in human beings this will be found intimately connected with the transfer by indirect generalization of the reactions from one stimulus pattern to another through the mediation of words. At very high levels of adaptive efficiency it is expected that words will constitute the mediating stimuli of the stimulus patterns themselves.

SUMMARY

Many life situations require for optimal chance of survival that the organism shall react to certain combinations of conditions (stimulus compounds) differently than to the component conditions, either when these components are encountered "separately" or in other combinations. The most radical, and at the same time the most simple, formulation of this problem is presented by Pavlov's experimental arrangement for the discrimination of a stimulus compound from its components. Experiments have fully demonstrated that organisms over a wide phylogenetic range are able to learn such discriminations, though usually with comparative difficulty.

This type of learning by organisms turns out upon analysis definitely to be a derived or secondary phenomenon, dependent upon a number of logically prior principles all of which have been recognized by Pavlov. Among the more important of these are the connection of the reaction (*R*) to the afferent impulse (*s*) set in motion by the stimulus (*S*), rather than directly to the stimulus;

the mutual interaction of afferent impulses; and the downward slope of the gradient of generalization both for excitation and for inhibition. For the derivation of temporal stimulus patterning there is required, in addition, the principle of the perseverative stimulus trace.

When stripped of quantitative details, the basic logic of stimulus patterning is rather simple. The afferent impulses produced by the components of a dynamic stimulus compound are to some extent different when the component is acting "alone," i.e., in a relatively static combination, than when it is acting with the remainder of the dynamic compound. If a reaction is conditioned to the compound, the reaction potential of a given component, because of the generalization gradient, is less when it is acting separately than when in the compound. During the differential reinforcement which produces this kind of learning, the generalized excitatory potential of the components is extinguished, developing inhibition in proportion to the reaction strength of each component. This inhibitory potential generalizes back upon the compound but, again, with a reduction due to the generalization gradient. The resulting net loss to the reaction potential at the command of the stimulus compound is much less than its original reaction potential; this ordinarily leaves the stimulus compound an amount of effective reaction potential which is well above the reaction threshold. This difference is the basis of the discrimination, i.e., of successful patterning. While the details of positive and negative simultaneous stimulus patterning and of positive and negative temporal stimulus patterning differ slightly, they all conform in substance to the summary account just given. The process of genuine stimulus patterning thus turns out to be at bottom the learning to discriminate afferent interaction effects.

By an analogous use of the same set of postulates it is possible also to deduce the phenomenon not only of genuine stimulus patterning, but also of quasi or spontaneous stimulus patterning, and, other things equal, the amount of both kinds of patterning as an increasing function of the degree of the afferent interaction effects mutually generated by the components. Other deductions are to the effect that positive patterning will be easier to learn than negative, that simultaneous compounds will be easier to pattern than temporal, and that the longer the time interval separating the components in a temporal stimulus compound, the more difficult will be the patterning. The same postulates afford a rather detailed

explanation of why a musical note to which a reaction has been conditioned fails to evoke the reaction when given in the midst of an arpeggio, the remaining notes of which have been made negative by differential reinforcement. All of these deductions are in substantial agreement with such empirical observations as are at present available.

If the organism could be certain of the occurrence of primary receptor discharges corresponding to every relevant condition in its environment which contributes to the determination of whether a given response will be followed by reinforcement, all responses in high-grade organisms might ultimately be made only to patterned stimulus compounds. But since under life conditions many elements which determine reinforcement do not activate any receptor, the basis for complete and exact patterning is frequently lacking; the organism must accordingly gamble on the outcome, often with its very life at stake. However, the processes of biological evolution seem to have produced a fairly satisfactory non-patterned arrangement for meeting this contingency.

Analysis suggests that the magnitude of ordinary neural interaction effects is such as to produce a fall of less than 50 per cent in the generalization gradient. Under these conditions the afferent impulse arising from a stimulus aggregate will tend to evoke the same reaction both alone and within the compound. When in the compound, the habit strengths (or reaction potentials) commanded by different stimulus aggregates presumably summate by a kind of diminishing returns principle. As a consequence, and quite apart from any patterning, the fewer the stimulus elements conditioned to a given reaction which chance to be present in a given stimulus compound, the smaller the reaction potentiality will be. This is believed to be a kind of crude but automatic biological calculus of the probability that a reaction evoked under given circumstances will be followed by reinforcement.

On the basis of the foregoing considerations we now formulate two important corollaries:

MAJOR COROLLARY IV

Differential reinforcement applied to simultaneous stimulus compounds results in the patterning of such compounds, either positively or negatively according to whether the compound or the components are reinforced.

MAJOR COROLLARY V

Differential reinforcement applied to temporal stimulus compounds results in the patterning of such compounds, either positively or negatively according to whether the compound or the components are reinforced.

NOTES

The Formula for Calculating the Empirical Degree of Patterning in the Case of Reactions Which Are Not of the All-or-None Type

The formula for P (Principle 2), while applying fairly well to reactions of the all-or-none type, definitely does not apply to a response such as the galvanic skin reaction, or the salivary reaction, whose amplitude varies with the magnitude of the reaction potential. A distinctly unsuccessful attempt at a formula for this latter type of reaction tried out in an earlier study (4) was,

$$P = \frac{R_1 + R_2}{R_1 + 2}$$

where R_1 is the amplitude of the reaction evoked by one stimulus component, and R_2 is the amplitude of that evoked by a second. Unfortunately, the determination of a suitable formula for the calculation of the extent of empirical patterning with this type of reaction requires a knowledge of the physiological limit of the amplitude of its conditioned evocation; to find this would probably be a very laborious procedure (4, p. 108 ff.).

The Patterning of Stimulus Compounds and the Configuration Psychologies

After studying the above chapter the reader may naturally ask what the relation of the present behavioristic treatment of the configurational problem in learning is to that put forward by the Wertheimer branch of the *Gestalt* school. While much might be said on this subject, the few words possible to devote to it in this place may help to clarify the reader's understanding.

Gestalt Theorie asserts that configurations are not only logically primary but that they are somehow primordial. Indeed, if current configurationism is ever formulated as a true scientific theory, so that its primary and secondary principles can be clearly distinguished, it is rather likely that a statement asserting the reality and nature of configurations will be revealed as its sole primary principle or postulate. The present work, on the other hand, undertakes to demonstrate that the response of organisms to stimulus configurations is logically secondary, that it is the result of a rather complex process of learning which is mediated by the behaviorally primary processes of (1) afferent neural interaction, (2) perseverative stimulus traces, (3) reinforcement, (4) generalization of reaction potential, (5) experimental extinction, and (6) generalization of inhibition.

Gestalt writers frequently leave the impression that an adequate derivation of the reaction of organisms to stimulus configurations is *a priori* impossible from behavioristic or non-consciousness principles. The position of the present work is that such a derivation is not only possible, but relatively simple and straightforward. Moreover, the preceding pages have presented a number of such deductions, thereby showing the *Gestalt a priori* claims to be mistaken.

Meanwhile it remains to be seen whether *Gestalt Theorie* can itself mediate comparable deductions. Clearly, no dispute exists as to the genuineness or the importance of the patterning of stimulus compounds; the difference of opinion concerns, rather, the logical question of whether stimulus patterning is a primary or a secondary principle. There are, of course, other differences between *Gestalt Theorie* and the present approach, but they do not particularly concern us here.

It is hoped that the derivation of the major phenomena of stimulus-pattern learning from objective, non-consciousness principles as demonstrated above, will contribute to the dissipation of current misunderstandings among psychologists, since these are a source of such deep and painful confusion to the scientific public. However, optimism in this connection is seriously dampened by the conviction that the differences involved arise largely from a conflict of cultures (6, pp. 18, 685; 7, p. 30) which, unfortunately, are extra-scientific and are not ordinarily resolvable by either logical or empirical procedures.

REFERENCES

1. BRUNSWIK, E. Organismic achievement and environmental probability. *Psychol. Rev.*, 1943, 50, 255-272.
2. HULL, C. L. A functional interpretation of the conditioned reflex. *Psychol. Rev.*, 1929, 36, 498-511.
3. HULL, C. L. Mind, mechanism, and adaptive behavior. *Psychol. Rev.*, 1937, 44, 1-32.
4. HULL, C. L. Explorations in the patterning of stimuli conditioned to the G.S.R. *J. Exper. Psychol.*, 1940, 27, 95-110.
5. HUMPHREY, G. *The nature of learning*. New York: Harcourt, Brace and Co., 1933.
6. KOFFKA, K. *Principles of Gestalt psychology*. New York: Harcourt, Brace and Co., 1935.
7. KÖHLER, W. *Gestalt psychology*. New York: Liveright Pub. Co., 1929.
8. PAVLOV, I. P. *Conditioned reflexes* (trans. by G. V. Anrep). London: Oxford Univ. Press, 1927.
9. RAZRAN, G. H. S. Studies in configural conditioning: V. Generalization and transposition. *J. Genet. Psychol.*, 1940, 56, 3-11.
10. WOODBURY, C. B. The learning of stimulus patterns by dogs. *J. Comp. Psychol.*, 1943, 35, 29-40.

CHAPTER XX

General Summary and Conclusions

In the foregoing chapters we have made a detailed examination of much experimental evidence, and we have considered the merits of many alternative interpretations. Such complications, while unavoidable in a work of this kind, necessarily tend to obscure an integrated view in which the various components of the subject have their proper significance. No doubt the reader has sighed more than once for the simplicity of dogmatic affirmation and for the over-all perspective attainable by brevity. In the present chapter we shall endeavor to make a clarifying integration of the major conclusions scattered through the preceding exposition.

THE NATURE OF SCIENTIFIC THEORY

The major task of science is the isolation of principles which shall be of as general validity as possible. In the methodology whereby scientists have successfully sought this end, two procedures may be distinguished—the empirical and the theoretical. The empirical procedure consists primarily of observation, usually facilitated by experiment. The theoretical procedure, on the other hand, is essentially logical in nature; through its mediation, in conjunction with the employment of the empirical procedure, the range of validity of principles may be explored to an extent quite impossible by the empirical procedure alone. This is notably the case in situations where two or more supposed primary principles are presumably operative simultaneously. The logical procedure yields a statement of the outcome to be expected if the several principles are jointly active as formulated; by comparing deduced or theoretical conclusions with the observed empirical outcomes, it may be determined whether the principles are general enough to cover the situation in question.

Scientific theory in its ideal form consists of a hierarchy of logically deduced propositions which parallel all the observed empirical relationships composing a science. This logical structure is derived from a relatively small number of self-consistent primary principles called postulates, when taken in conjunction with relevant

antecedent conditions. The behavior sciences have been slower than the physical sciences to attain this systematic status, in part because of their inherent complexity, in part because of the action of the oscillation principle, but also in part because of the greater persistence of anthropomorphism.

Empirical observation, supplemented by shrewd conjecture, is the main source of the primary principles or postulates of a science. Such formulations, when taken in various combinations together with relevant antecedent conditions, yield inferences or theorems, of which some may agree with the empirical outcome of the conditions in question, and some may not. Primary propositions yielding logical deductions which consistently agree with the observed empirical outcome are retained, whereas those which disagree are rejected or modified. As the sifting of this trial-and-error process continues, there gradually emerges a limited series of primary principles whose joint implications are progressively more likely to agree with relevant observations. Deductions made from these surviving postulates, while never absolutely certain, do at length become highly trustworthy. This is in fact the present status of the primary principles of the major physical sciences.

BEHAVIOR THEORY AND SYMBOLIC CONSTRUCTS

Scientific theories are mainly concerned with dynamic situations, i.e., with the consequent events or conditions which, with the passage of time, will follow from a given set of antecedent events or conditions. The concrete activity of theorizing consists in the manipulation of a limited set of symbols according to the rules expressed in the postulates (together with certain additional rules which make up the main substance of logic) in such a way as to span the gap separating the antecedent conditions or states from the subsequent ones. Some of the symbols represent observable and measurable elements or aggregates of the situation, whereas others represent presumptive intervening processes not directly subject to observation. The latter are theoretical constructs. All well-developed sciences freely employ theoretical constructs wherever they prove useful, sometimes even sequences or chains of them. The scientific utility of logical constructs consists in the mediation of valid deductions; this in turn is absolutely dependent upon every construct, or construct chain, being securely anchored both on the antecedent and on the consequent side to conditions or events

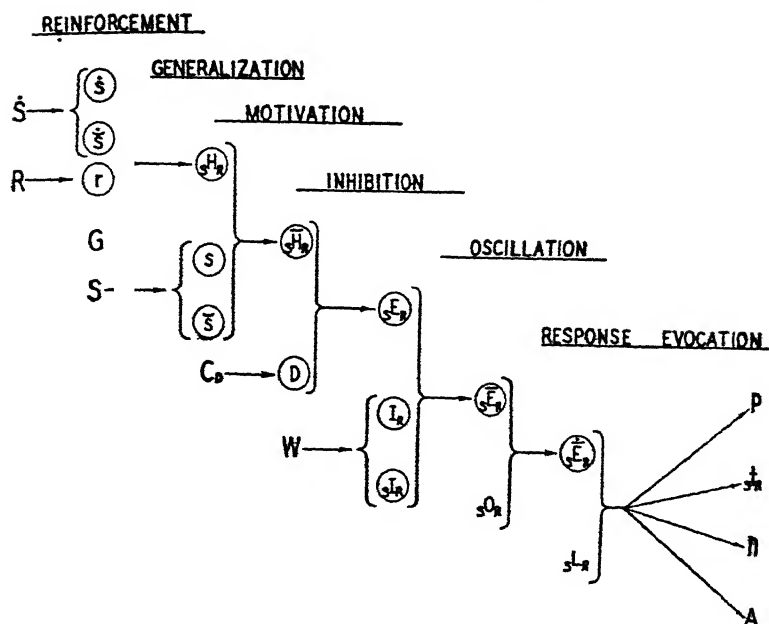


FIG. 84. Diagram summarizing the major symbolic constructs (encircled symbols) employed in the present system of behavior theory, together with the symbols of the supporting objectively observable conditions and events. In this diagram \dot{S} represents the physical stimulus energy involved in learning; R , the organism's reaction; \hat{s} , the neural result of the stimulus; \hat{s} , the neural interaction arising from the impact of two or more stimulus components; r , the efferent impulse leading to reaction; G , the occurrence of a reinforcing state of affairs; H_x , habit strength; S , evocation stimulus on the same stimulus continuum as \dot{S} ; \bar{H}_x , the generalized habit strength; C_D , the objectively observable phenomena determining the drive; D , the physiological strength of the drive to motivate action; \bar{E}_x , the reaction potential; W , work involved in an evoked reaction; I_x , reactive inhibition; \bar{I}_x , conditioned inhibition; \bar{E}_x , effective reaction potential; O_x , oscillation; \bar{E}_x , momentary effective reaction potential; L_x , reaction threshold; p , probability of reaction evocation; t_x , latency of reaction evocation; n , number of unreinforced reactions to produce experimental extinction; and A , amplitude of reaction. Above the symbols the lines beneath the words *reinforcement*, *generalization*, *motivation*, *inhibition*, *oscillation*, and *response evocation* indicate roughly the segments of the chain of symbolic constructs with which each process is especially concerned.

which are directly observable. If possible, they should also be measurable.

The theory of behavior seems to require the use of a number of symbolic constructs, arranged for the most part in a single chain. The main links of this chain are represented in Figure 84.

In the interest of clarity, the symbolic constructs are accompanied by the more important and relevant symbols representing the objectively anchoring conditions or events. In order that the two types of symbols shall be easily distinguishable, circles have been drawn around the symbolic constructs. It will be noticed that the symbols representing observables, while scattered throughout the sequence, are conspicuously clustered at the beginning and at the end of the chain, where they must be in order to make validation of the constructs possible. Frequent reference will be made to this summarizing diagram throughout the present chapter, as it reveals at a glance the groundwork of the present approach to the behavior sciences.

ORGANISMS CONCEIVED AS SELF-MAINTAINING MECHANISMS

From the point of view of biological evolution, organisms are more or less successfully self-maintaining mechanisms. In the present context *a mechanism is defined as a physical aggregate whose behavior occurs under ascertainable conditions according to definitely statable rules or laws.* In biology, the nature of these aggregates is such that for individuals and species to survive, certain optimal conditions must be approximated. When conditions deviate from the optimum, equilibrium may as a rule be restored by some sort of action on the part of the organism; such activity is described as "adaptive." The organs effecting the adaptive activity of animals are for the most part glands and muscles.

In higher organisms the number, variety, and complexity of the acts required for protracted survival is exceedingly great. The nature of the act or action sequence necessary to bring about optimal conditions in a given situation depends jointly (1) upon the state of disequilibrium or need of the organism and (2) upon the characteristics of the environment, external and internal. For this reason a prerequisite of truly adaptive action is that both the condition of the organism and that of all relevant portions of the environment must somehow be brought simultaneously to bear on the reactive organs. The first link of this necessary functional *rapport* of the effector organs with organismic needs and environmental conditions is constituted by receptors which convert the biologically more important of the environmental energies (*S*) into neural impulses (*s*). For the most part these neural impulses flow to the brain, which acts as a kind of automatic switchboard medi-

ating their efferent flow (r) to the effectors in such a way as to evoke response (R). In this connection there are two important neural principles to be noted.

The first of these principles to be observed is that after the stimulus (S) has ceased to act upon the receptor, the afferent impulse (s) continues its activity for some seconds, or possibly minutes under certain circumstances, though with gradually decreasing intensity. This *perseverative stimulus trace* is biologically important because it brings the effector organ *en rapport* not only with environmental events which are occurring at the time but with events which have occurred in the recent past, a matter frequently critical for survival. Thus is effected a short-range temporal integration (Postulate 1, p. 47).

The second neural principle is that the receptor discharges and their perseverative traces (s) generated on the different occasions of the impact of a given stimulus energy (S) upon the receptor, while usually very similar, are believed almost never to be exactly the same. This lack of uniformity is postulated as due (1) to the fact that many receptors are activated by stimulus energies simultaneously and (2) to "afferent neural interaction." The latter hypothesis states that the receptor discharges interact, while passing through the nervous system to the point where newly acquired receptor-effector connections have their locus, in such a way that each receptor discharge changes all the others to a greater or less extent; i.e., s is changed to \check{s}_1 , \check{s}_2 , or \check{s}_3 , etc., in accordance with the particular combination of other stimulus energies which is acting on the sensorium at the time (see Figure 84). This type of action is particularly important because the mediation of the responses of organisms to distinctive combinations or patterns of stimuli, rather than to the components of the patterns, is presumably dependent upon it (Postulate 2, p. 47).

The detailed physiological principles whereby the nervous system mediates the behavioral adaptation of the organism are as yet far from completely known. As a result we are forced for the most part to get along as best we can with relatively coarse molar formulations derived from conditioned-reflex and other behavior experiments. From this point of view it appears that the processes of organic evolution have yielded two distinct but closely related means of effective behavioral adaptation. One of these is the laying down of unlearned receptor-effector connections (sU_R) within the neural tissue which will directly mediate at least approximate behavioral

adjustments to urgent situations which are of frequent occurrence but which require relatively simple responses (Postulate 3, p. 66). The second means of effecting behavioral adjustment is probably evolution's most impressive achievement; this is the capacity of organisms themselves to acquire automatically adaptive receptor-effector connections. Such acquisition is *learning*.

LEARNING AND THE PROBLEM OF REINFORCEMENT

The substance of the elementary learning process as revealed by much experimentation seems to be this: A condition of need exists in a more or less complex setting of receptor discharges initiated by the action of environmental stimulus energies. This combination of circumstances activates numerous vaguely adaptive reaction potentials mediated by the unlearned receptor-effector organization (sU_R) laid down by organic evolution. The relative strengths of these various reaction potentials are varied from instant to instant by the oscillation factor (sO_R). The resulting spontaneous variability of the momentary unlearned reaction potential (sU_R) produces the randomness and variability of the unlearned behavior evoked under given conditions. In case one of these random responses, or a sequence of them, results in the reduction of a need dominant at the time, there follows as an indirect effect what is known as reinforcement (G , of Figure 84). This consists in (1) a strengthening of the particular receptor-effector connections which originally mediated the reaction and (2) a tendency for all receptor discharges (s) occurring at about the same time to acquire new connections with the effectors mediating the response in question. The first effect is known as primitive trial-and-error learning; the second is known as conditioned-reflex learning. In most adaptive situations both processes occur concurrently; indeed, *very likely they are at bottom the same process, differing only in the accidental circumstance that the first begins with an appreciable strength, whereas the second sets out from zero.* As a result, when the same need again arises in this or a similar situation, the stimuli will activate the same effectors more certainly, more promptly, and more vigorously than on the first occasion. Such action, while by no means adaptively infallible, in the long run will reduce the need more surely than would a chance sampling of the unlearned response tendencies (sU_R) at the command of other need and stimulating situations, and more quickly

and completely than did that particular need and stimulating situation on the first occasion. Thus the acquisition of such receptor-effector connections will, as a rule, make for survival; i.e., it will be adaptive.

Careful observation and experiment reveal, particularly with the higher organisms, large numbers of situations in which learning occurs with no associated primary need reduction. When these cases are carefully studied it is found that the reinforcing agent is a situation or event involving a stimulus aggregate or compound which has been closely and consistently associated with the need reduction. Such a situation is called a secondary reinforcing agent, and the strengthening of the receptor-effector connections which results from its action is known as secondary reinforcement. This principle is of immense importance in the behavior of the higher species.

The organization within the nervous system brought about by a particular reinforcement is known as a habit; since it is not directly observable, habit has the status of a symbolic construct. Strictly speaking, habit is a functional connection between s and r ; it is accordingly represented by the symbol ${}_sH_r$. Owing, however, to the close functional relationship between S and s on the one hand, and between r and R on the other, the symbol ${}_sH_R$ will serve for most expository purposes; the latter symbol has the advantage that S and R both refer to conditions or events normally open to public observation. The position of ${}_sH_R$ in the chain of constructs of the present system is shown in Figure 84.

While it is difficult to determine the quantitative value of an unobservable, various indirect considerations combine to indicate as a first approximation that habit strength is a simple increasing growth function of the number of reinforcements. The unit chosen for the expression of habit strength is called the *hab*, a shortened form of the word "habit"; a *hab* is 1 per cent of the physiological limit of habit strength under completely optimal conditions.

CONDITIONS WHICH INFLUENCE THE MAGNITUDE OF HABIT INCREMENT PER REINFORCEMENT

A more careful scrutiny of the conditions of reinforcement reveals a number which are subject to variation, and experiments have shown that the magnitude of the habit increment ($\Delta {}_sH_R$) per reinforcement is dependent in one way or another upon the

quantitative variation of these conditions. One such factor concerns the primary reinforcing agent. It has been found that, quality remaining constant, the magnitude of the increment of habit strength per reinforcement is a negatively accelerated increasing function of the quantity of the reinforcing agent employed per reinforcement.

A second factor of considerable importance in determining the magnitude of $\Delta_s H_R$ is the degree of asynchronism between the onset of the stimulus and of the response to which it is being conditioned. This situation is complicated by whether or not the stimulus terminates its action on the receptor before the response occurs. In general the experimental evidence indicates that in case both the stimulus and the response are of very brief duration, the increment of habit strength per reinforcement is maximal when the reaction (and the reinforcement) occurs a short half second after the stimulus, and that it is a negatively accelerated decreasing function of the extent to which asynchronisms in either direction depart from this optimum. In case the reaction synchronizes with the continued action of the stimulus on the receptor, the increment of habit strength per reinforcement is a simple negative growth function of the length of time that the stimulus has acted on the receptor when the reaction occurs.

A third important factor in the reinforcing situation is the length of time elapsing between the occurrence of the reaction and of the reinforcing state of affairs (G , Figure 84). Experiments indicate that this "gradient of reinforcement" is a negatively accelerated decreasing growth function of the length of time that reinforcement follows the reaction. The principle of secondary reinforcement, combined with that of the gradient of reinforcement, explains the extremely numerous cases of learning in which the primary reinforcement is indefinitely remote from the act reinforced. A considerable mass of experimental evidence indicates that a kind of blending of the action of these two principles generates a secondary phenomenon called the "goal gradient." Upon empirical investigation this turns out to be a decreasing exponential or negative growth function of the time (t) separating the reaction from the primary reinforcement for delays ranging from ten seconds to five or six minutes; delays greater than six minutes have not yet been sufficiently explored to make possible a quantitative statement concerning them.

There are doubtless other conditions which influence the magni-

tude of the increment of habit strength resulting from each reinforcement. Those listed above certainly are typical and probably comprise the more important of them. An adequate statement of the primary law or laws of learning would accordingly take the form of an equation in which ${}_sH_R$ would be expressed as a joint function not only of N but of the quantity and quality of the reinforcing agent, and of the temporal relationships of S to R and of R to G . A formula which purports to be a first approximation to such a general quantitative expression of the primary laws of learning is given as equations 16 and 17, pp. 178-179.

STIMULUS GENERALIZATION

With the primary laws of learning formally disposed of, we proceed to the consideration of certain dynamical principles according to which habits, in conjunction with adequate stimulation (S) and drive (D), mediate overt behavior. In this connection we note the fact that a stimulus (S , Figure 84), through its afferent impulses (s , represented in Figure 84) will often evoke the reaction (R) even though s may be rather different from \dot{s} or \ddot{s} , the receptor impulse originally conditioned to R . This means that when a stimulus (\dot{S}) and a reaction (R) are conjoined in a reinforcement situation, there is set up a connection not only to the stimulus involved in the reinforcement but to a whole zone of other potential stimuli lying on the same stimulus continuum, such as S_1 , S_2 , S_3 , and so forth. This fact, known as stimulus generalization, is of immense adaptive significance; since stimuli are rarely if ever exactly repeated, habits could scarcely function adaptively without it.

Stimulus generalization has the characteristic that in general the greater the physical deviation of S from \dot{S} , the weaker will be the habit strength which is mobilized. More precisely, the strength of a generalized habit (${}_s\bar{H}_R$) is a linear increasing function of the strength of the habit at the point of reinforcement and a negatively accelerated decreasing function of the difference (d) between \dot{S} and S as measured in discrimination thresholds (j.n.d.'s). Thus ${}_s\bar{H}_R$ is a theoretical construct anchored to the construct ${}_sH_R$ and to the observables \dot{S} and S (see Figure 84).

Stimulus generalization appears to take two forms—(1) qualitative stimulus generalization and (2) stimulus intensity generalization; another way of stating the same thing is to say that each

more rapid than is the ordinary loss of learning effects. This would produce the phenomenon of reminiscence which has been especially studied in rote learning.

Since the presence of I_R constitutes a need, the cessation of the activity which generated the need would initiate the need-reduction process; but since need reduction is the critical element in reinforcement, there follows with fair plausibility the molar principle that cessation of the activity would be conditioned to any stimuli which are consistently associated with such cessation (Postulate 9, p. 300). But a tendency to the cessation of an act would be directly inhibitory to the performance of that act. Therefore the inclusion of such an inhibitory stimulus in a stimulus compound, the remainder of which is positively conditioned to the response, would tend to prevent the evocation of the response in question; this is, in fact, the ordinary empirical test for conditioned inhibition (sI_R , Figure 84).

On the above view that sI_R is a negative habit, the injection of alien stimuli into the stimulus compound would, through the principle of afferent interaction, produce disinhibition, i.e., a temporary reduction or total abolition of sI_R . But on the assumption that sI_R is being set up during the process of accumulating I_R , it follows that the total inhibition (I_R) at the conclusion of experimental extinction must be in part I_R and in part sI_R . For this reason disinhibition will take place only in so far as I_R is composed of sI_R , and spontaneous recovery will take place only in so far as I_R is composed of I_R ; this means that neither disinhibition nor spontaneous recovery can ever restore an extinguished reaction potential to its full original strength. Other implications which flow from the above assumptions are that there is greater economy in distributed than in massed repetitions in rote learning, and that, other things equal, organisms receiving the same reinforcement following two responses which require different energy expenditures will, as practice continues, gradually come to choose the less laborious response. This is the "law of less work."

Implicit in the preceding discussion has been the assumption that the reaction potential actually available for reaction evocation, i.e., the effective reaction potential ($s\bar{E}_R$, Figure 84), is what remains of the reaction potential (sE_R) after the subtraction of the total inhibition, I_R ; i.e.,

$$s\bar{E}_R = sE_R - I_R.$$

Since both ${}_sE_R$ and \dot{I}_R are anchored to objectively observable antecedent conditions, it follows that ${}_s\overline{E}_R$ is also thus anchored.

THE OSCILLATION OF EFFECTIVE REACTION POTENTIAL

At this point it must be noted at once that the full value of ${}_s\overline{E}_R$ is rarely brought to bear in the evocation of action. Instead it is subject to random or chance downward variability. These fluctuations are believed to be due to a little-understood physiological process which has the power of neutralizing reaction potentials to degrees varying from moment to moment. Because of this latter characteristic, the process is called "oscillation"; it is represented by the symbol ${}_sO_R$. Effective reaction potential as modified by oscillation is called "momentary effective reaction potential"; this is represented by the symbol ${}_s\overline{E}_R$.

Since ${}_sO_R$ is not directly observable, it has something of the status of a symbolic construct; on the other hand, owing to its presumably constant value, it has less elusiveness than an ordinary construct; it is therefore not placed in a circle in Figure 84. The hypothetical characteristics of ${}_sO_R$ may be listed as follows:

1. It is active at all times.
2. It exerts an absolute depressing action against any and all reaction potentials, whether great or small.
3. The magnitude of this potentiality varies from instant to instant according to the normal probability distribution.
4. The magnitude of its action on different reaction potentials at a given instant is uncorrelated (Postulate 10, p. 319).

Since oscillation is continuously active on all reaction potentials, it plays a very great rôle in adaptive behavior. It presumably is responsible for many of the phenomena grouped by the classical psychologists under the head of "attention." It is in large measure responsible for the fact that the social sciences must pool many observations before ordinary empirical laws may become manifest. Thus natural laws in the social sciences must always be based on statistical indices of one kind or another. This in its turn has induced much preoccupation with statistical methods on the part of the various behavior sciences. The necessity of pooling large numbers of observations in order to isolate empirical laws has greatly increased the labor associated with empirical investigations and has doubtless appreciably retarded the development of the behavior sciences.

THE REACTION THRESHOLD AND RESPONSE EVOCATION

The anchoring on the posterior or consequent side of our chain of behavioral constructs culminating in $s\bar{E}_R$, as shown in Figure 84, lies in the evocation of observable reactions. In the determination of the functional relationship of $s\bar{E}_R$ to the various measurable phenomena of responses, we encounter special difficulties owing to the fact that $s\bar{E}_R$ is itself not directly observable. If we were quite sure of the quantitative functional relationship of $s\bar{E}_R$ to its combination of antecedent anchors, the value of $s\bar{E}_R$ could be calculated in empirical situations and equations could then be fitted to the relationship of these numbers to the corresponding response values; these equations are what we seek. Unfortunately the necessary antecedent functional relationships are not yet known with sufficient certainty.

It happens, however, that in typical sets of simple learning results, employing the four measurable response phenomena, the fitted equations in all cases are easily and naturally expressible by equations involving the simple positive growth (exponential) function of the number of reinforcements (N). This tends somewhat to confirm the soundness of the general growth hypothesis of the relation of N to sH_R , and so of N to $s\bar{E}_R$. Further independent confirmation of the soundness of the growth hypothesis of the relation of $s\bar{E}_R$ to N , lies in the following fact: when the probability-of-reaction-evocation type of learning curve is analyzed theoretically, it turns out to be yielded in a degree of detail scarcely attributable to chance on the above assumption of the relation of $s\bar{E}_R$ to N coupled with two additional assumptions, each well supported by independent evidence—that of the oscillation function (sO_R) and that of the reaction threshold (sL_R) (see Figure 84). The characteristics of the oscillation function have been summarized above. Moreover, the concept of the reaction threshold is well established, since notions fairly comparable to it have long been current in classical psychophysics and in physiology. As here employed, the reaction threshold (sL_R) is the minimal amount of momentary effective reaction potential ($s\bar{E}_R$) which is necessary to mediate reaction evocation when the situation is uncomplicated by competing reaction potentials (Postulate 11, p. 344).

Acting, then, on the fairly well-authenticated growth hypothesis of the relation of $s\bar{E}_R$ to N , it is a relatively simple matter, by

inspecting the equations fitted to concrete examples of the three remaining types of learning curves and utilizing the method of residues, to determine the functional relationship of $s\bar{E}_R$ to the particular behavior phenomena employed. As a result of this procedure it is concluded that probability of reaction evocation stands in an ogival relationship to effective reaction potential (Postulate 12, p. 344); that reaction latency stands in a negatively accelerated inverse relationship (Postulate 13, p. 344); and that both resistance to experimental extinction and reaction amplitude (of autonomically mediated responses) are increasing linear functions of $s\bar{E}_R$ (Postulates 14 and 15, p. 344).

A final complication concerning reaction evocation arises from the fact that often the stimulus elements impinging on the receptors at a given instant may mobilize superthreshold reaction potentials to several different reactions, some or all of which may be mutually incompatible. In such cases all but the strongest will necessarily suffer associative inhibition (Postulate 16, p. 344). There are also some indications that the dominant potential itself may suffer a certain amount of blocking; indeed, this is the basis of the most plausible theory of "forgetting" now available.

This concludes our summary of primary principles. All of these principles are also statable in the form of quantitative equations. This means that if the antecedent conditions \hat{S} , \hat{s} , R , G , t , t' , S , \check{s} , C_D , W , sO_R , and sL_R were known, it would be possible to compute p , st_R , n , or A by substituting appropriately in a succession of these equations beginning on the left-hand side of Figure 84 and proceeding toward the right. For example, the calculation of st_R would employ equations 16, 34, 44, 45, and 48.

DYNAMICS OF STIMULUS COMPOUNDS AND PATTERNS

For the most part the molar principles outlined in the preceding chapters are presumably primary in nature, though occasional secondary principles have been presented. Because of their relatively primitive status in the logical hierarchy of the system and of their especially intimate relation to survival, a few secondary principles or mechanisms have been given special consideration and have been listed as "major corollaries." One of these concerns the quantitative summation of the reaction potentials mobilized by the several stimulus components of a stimulus compound, and the

other concerns the matter of stimulus patterning. We shall first take up the matter of the summation of reaction potentials.

In spite of the presumptive fact of afferent neural interaction, the afferent discharge of each receptor contains a large amount of similarity regardless of the influence of other stimulus elements (and receptors) which may be active at the time. This means that any stimulus component conditioned to a reaction will ordinarily command an appreciable potentiality to that reaction regardless of the other stimuli accompanying it. Now, according to the primary law of learning, each individual receptor discharge bears its load of habit strength, and so of reaction potential. The reaction-potential loadings thus borne by the several receptor discharges initiated by the different stimulus elements of a stimulus compound presumably combine quantitatively in the same way as do the different increments of habit strength, i.e., not by a simple addition but according to a kind of diminishing-returns principle. Thus if two stimulus aggregates bearing equal loads of reaction potential to the evocation of the same response are acting simultaneously as a stimulus compound, their physiological summation, quite apart from afferent interaction effects, will be less than the arithmetical sum of the two reaction potentials; similarly, if one of the two equally loaded stimulus aggregates making up a stimulus compound should be withdrawn from the compound, more than half of the total reaction potential would remain. As a result (except for afferent neural interaction effects), the more completely a reinforced stimulus compound is repeated on a subsequent occasion, the more likely it will be to evoke the reaction in question.

This mode of action has special adaptive significance, because the more completely the stimulus compound is repeated, the more similar will be the environmental situation in general to the situation in which need reduction originally occurred, and therefore the more probably will the response in question lead again to a reduction in the need. Here we have a primitive automatic mechanism which in effect roughly gauges the probability of a given stimulus situation's yielding need reduction in case a given response is evoked. This adaptive mechanism has the great advantage of being instantly available at the presentation of any stimulus situation, novel or otherwise.

The other secondary principle mediating the response of organisms to stimulus compounds, which we have included in the present work, is that known as patterning. This operates concurrently

with the summation principle just discussed but is much slower in its action. However, given sufficient time for the rather difficult learning process to take place, stimulus patterning may be very precisely adaptive. It is a fact that in very large numbers of situations the question of whether or not a given response will be followed by reinforcement depends upon the presence or absence of a particular combination of physical circumstances and so, for the organism, upon a particular combination or pattern of stimulus elements, rather than upon the presence or absence of any of the components. Since each combination of stimulus elements will modify to some extent the afferent impulses produced by each stimulus component, any change in the stimulus compound will also modify to some extent the afferent responses initiated by all the remaining stimulus components. In the process of the irregular alternation of reinforcement and extinction called differential reinforcement, which is characteristic of the form of trial and error known as discrimination learning, higher organisms are able to emerge with one response successfully conditioned to one combination of stimuli and with a quite different response successfully conditioned to another combination of stimuli containing many of the components of the first, provided some of the elements are different. At bottom this discrimination is possible because the afferent impulse \check{s}_1 which arises from the stimulus element S_1 when occurring concurrently with the stimulus element S_2 , is to some extent different from \check{s}_3 , which arises from the same stimulus element, S_1 , when occurring concurrently with a different stimulus element, S_3 . The physiological summation of the several component reaction potentials characteristic of various stimulus patterns which have many, and even most, of their stimulus elements in common, accordingly may result in the evocation without confusion of the distinctive reaction conditioned to each. Thus each of the forty or so elementary speech sounds is a fairly distinctive pattern made up of a "fundamental" physical vibration rate and a particular combination of higher partials. Each of the thousands of words of the better-developed languages consists of a temporally patterned sequence of these elementary speech sounds, stops, and so forth. In reading, each letter is a complex visual pattern, each word is a complex pattern of these letter patterns, and each sentence is a temporally patterned sequence of printed word patterns. Indeed, it is impossible to think of a life situation which is not patterned to a considerable extent. The limiting case of this kind of

learning is that in which a stimulus compound is conditioned to evoke a reaction while the several components when acting alone are consistently extinguished.

A FORWARD GLANCE

The main concern of this work has been to isolate and present the primary or basic principles or laws of behavior as they appear in the current state of behavioral knowledge; at present there have been isolated sixteen such principles. In so far as these principles or postulates are sound and sufficient, it should be possible to deduce from them an extensive logical hierarchy of secondary principles which will exactly parallel all of the objectively observable phenomena of the behavior of higher organisms; such a hierarchy would constitute a systematic theory of all the social sciences. Considerable progress has been made in this direction (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 22, 23, 24, 25, 26, 27, 28, 29), though because of the limitations in available space only a random sampling of some fifty or so secondary principles (corollaries) is included in the present volume; these are given chiefly for purposes of illustrating the meaning of the primary principles.

As the systematization of the behavior sciences proceeds, some of the principles put forward above as primary will be found to yield false deductions and will therefore be abandoned; some will be discarded as primary principles because found derivable from other primary principles and consequently will be placed in the group of secondary principles; others will be found partially defective and will require modification; finally, entirely new postulates will need to be added. The primary principles presented in the preceding pages have been formulated with the certainty of these future developments fully in mind. A sharp and definite formulation has, in many cases, been given principles despite admitted doubt as to their precise validity. It is believed that a clear formulation, even if later found incorrect, will ultimately lead more quickly and easily to a correct formulation than will a pussyfooting statement which might be more difficult to convict of falsity. The primary task of a science is the early and economical discovery of its basic laws. In the view of the scientifically sophisticated, to make an incorrect guess whose error is easily detected should be no disgrace; scientific discovery is in part a trial-and-error process,

and such a process cannot occur without erroneous as well as successful trials. On the other hand, to employ a methodology by which it is impossible readily to detect a mistake once made, or deliberately to hide a possible mistake behind weasel words, philosophical fog, and anthropomorphic prejudice, slows the trial-and-error process, and so retards scientific progress.

It is to be hoped that as the years go by, systematic treatises on the different aspects of the behavior sciences will appear. One of the first of these would naturally present a general theory of individual behavior; another, a general theory of social behavior. In the elaboration of various subdivisions and combinations of these volumes there would develop a systematic series of theoretical works dealing with different specialized aspects of mammalian behavior, particularly the behavior of human organisms. Such a development would include volumes devoted to the theory of skills and their acquisition; of communicational symbolism or language (semantics); of the use of symbolism in individual problem solution involving thought and reasoning; of social or ritualistic symbolism; of economic values and valuation; of moral values and valuation; of aesthetic values and valuation; of familial behavior; of individual adaptive efficiency (intelligence); of the formal educative processes; of psychogenic disorders; of social control and delinquency; of character and personality; of culture and acculturation; of magic and religious practices; of custom, law, and jurisprudence; of politics and government; and of many other specialized behavior fields.

As a culmination of the whole there would finally appear a work consisting chiefly of mathematics and mathematical logic. This would set out with a list of undefined terms or signs whose referents are publicly available to the observation of all normal persons; such terms, because they can be directly conditioned to the referents by differential reinforcement, should have a minimum of ambiguity. From these undefined notions would be synthesized by the incomparable technique of symbolic logic all the critical concepts required by the system, for correct primary concepts are just as important for valid systematization in science as are correct primary principles; this should yield a complete set of wholly unambiguous terms. From these terms or signs would be formulated precise mathematical statements of the several postulates or primary molar principles which survive the intervening winnowing process, together with such other principles as it may be found

necessary to introduce; from these, by means of rigorous mathematical processes, would be derived theorems paralleling all the empirical ramifications of the so-called social sciences. Also there should be derivable large numbers of theorems concerning the outcome of situations never yet investigated; this latter group would make possible practical behavior applications and social inventions.

If one may judge by the history of the older sciences, it will be a long time before the "social" sciences attain a status closely approximating that contemplated here. Nevertheless there is reason to hope that the next hundred years will see an unprecedented development in this field. One reason for optimism in this respect lies in the increasing tendency, at least among Americans, to regard the "social" or behavioral sciences as genuine natural sciences rather than as *Geisteswissenschaft*. Closely allied to this tendency is the growing practice of excluding theological, folk, and anthropomorphic considerations from the list of the presumptive primary behavioral explanatory factors. Wholly congruent with these tendencies is the expanding recognition of the desirability in the behavior sciences of explicit and exact systematic formulation, with empirical verification at every possible point. If these three tendencies continue to increase, as seems likely, there is good reason to hope that the behavioral sciences will presently display a development comparable to that manifested by the physical sciences in the age of Copernicus, Kepler, Galileo, and Newton.

But we should not deceive ourselves. The task of systematically developing the behavior sciences will be both arduous and exacting, and many radical changes must occur. Behavior scientists must not only learn to read mathematics understandingly—they must learn to *think* in terms of equations and the higher mathematics. The so-called social sciences will no longer be a division of *belles lettres*; anthropomorphic intuition and a brilliant style, desirable as they are, will no longer suffice as in the days of William James and James Horton Cooley. Progress in this new era will consist in the laborious writing, one by one, of hundreds of equations; in the experimental determination, one by one, of hundreds of the empirical constants contained in the equations; in the devising of practically usable units in which to measure the quantities expressed by the equations; in the objective definition of hundreds of symbols appearing in the equations; in the rigorous deduction, one by one, of thousands of theorems and corollaries from the primary definitions and equations; in the meticulous per-

formance of thousands of critical quantitative experiments and field investigations designed with imagination, sagacity, and daring to test simultaneously the validity of both the theorems and the primary principles and concepts from which the former have been derived; in the ruthless discard or revision of once promising primary principles or concepts which have failed wholly or in part to meet the test of empirical validation.

There will be encountered vituperative opposition from those who cannot or will not think in terms of mathematics, from those who prefer to have their scientific pictures artistically out of focus, from those who are apprehensive of the ultimate exposure of certain personally cherished superstitions and magical practices, and from those who are associated with institutions whose vested interests may be fancied as endangered.

This great task can be no more than begun by the present generation of workers. Hope lies, as always, in the oncoming youth, those now in training and those to be trained in the future. Upon them rests the burden of the grinding and often thankless labor involved, and to them must rightfully go the thrill of intellectual adventure and the credit for scientific achievement. Perhaps they will have the satisfaction of creating a new and better world, one in which, among other things, there will be a really effective and universal moral education. The present work is primarily addressed to them.

REFERENCES

1. FORD, C. S. Culture and human behavior. *Scientific Monthly*, 1942, 55, 546-557.
2. GIBSON, E. J. A systematic application of the concepts of generalization and differentiation to verbal learning. *Psychol. Rev.*, 1940, 47, 196-229.
3. GUTHRIE, E. R. *The psychology of learning*. New York: Harper and Bros., 1935.
4. GUTHRIE, E. R. *The psychology of human conflict*. New York: Harper and Bros., 1938.
5. HILL, C. J. Goal gradient, anticipation, and perseveration in compound trial-and-error learning. *J. Exper. Psychol.*, 1930, 25, 566-585.
6. HOLT, E. B. *Animal drive and the learning process*. New York: Henry Holt and Co., 1931.
7. HULL, C. L. Simple trial-and-error learning: a study in psychological theory. *Psychol. Rev.*, 1930, 37, 241-256.
8. HULL, C. L. Knowledge and purpose as habit mechanisms. *Psychol. Rev.*, 1930, 37, 511-525.
9. HULL, C. L. Goal attraction and directing ideas conceived as habit phenomena. *Psychol. Rev.*, 1931, 38, 487-506.

10. HULL, C. L. The goal gradient hypothesis and maze learning. *Psychol. Rev.*, 1932, 39, 25-43.
11. HULL, C. L. The concept of the habit-family hierarchy and maze learning. *Psychol. Rev.*, 1934, 41, 33-52; 134-152.
12. HULL, C. L. The mechanism of the assembly of behavior segments in novel combinations suitable for problem solution. *Psychol. Rev.*, 1935, 42, 219-245.
13. HULL, C. L. Mind, mechanism, and adaptive behavior. *Psychol. Rev.*, 1937, 44, 1-32.
14. HULL, C. L. The goal-gradient hypothesis applied to some 'field-force' problems in the behavior of young children. *Psychol. Rev.*, 1938, 45, 271-299.
15. HULL, C. L. Simple trial-and-error learning—an empirical investigation. *J. Comp. Psychol.*, 1939, 27, 233-258.
16. HULL, C. L. *Psychology seminar memoranda, 1939-1940*. Bound mimeographed material on file in the libraries of the University of Iowa, Oberlin College, Yale University.
17. HULL, C. L. Conditioning: outline of a systematic theory of learning. Chapt. II in *The psychology of learning*, 41st Yearbook Natl. Soc. Study of Education, Part II. Bloomington, Ill.: Public School Pub. Co., 1942.
18. HULL, C. L., HOVLAND, C. I., ROSS, R. T., HALL, M., PERKINS, D. T., and FITCH, F. B. *Mathematico-deductive theory of rote learning*. New Haven: Yale Univ. Press, 1940.
19. HULL, C. L., and MOWBRER, O. H. *Hull's psychological seminars, 1936-38, Notices and abstracts of proceedings*. Bound mimeographed material on file in the libraries of the University of Chicago, University of North Carolina, and Yale University.
20. MILLER, N. E., and DOLLARD, J. *Social learning and imitation*. New Haven: Yale Univ. Press, 1941.
21. REICHE, F. *The quantum theory*. New York: E. P. Dutton, 1930.
22. ROUSE, R. O. The oscillation function in compound trial-and-error learning. *J. Comp. Psychol.*, 1943, 35, 177-186.
23. SPENCE, K. W. The nature of discrimination learning in animals. *Psychol. Rev.*, 1936, 43, 427-449.
24. SPENCE, K. W. The differential response in animals to stimuli varying within a single dimension. *Psychol. Rev.*, 1937, 44, 430-444.
25. SPENCE, K. W. Analysis of formation of visual discrimination habits in chimpanzee. *J. Comp. Psychol.*, 1937, 23, 77-100.
26. SPENCE, K. W. Continuous versus non-continuous interpretations of discrimination learning. *Psychol. Rev.*, 1940, 47, 271-288.
27. SPENCE, K. W. Failure of transposition in size-discrimination of chimpanzees. *Amer. J. Psychol.*, 1941, 54, 223-229.
28. SPENCE, K. W. The basis of solution by chimpanzees of the intermediate size problem. *J. Exper. Psychol.*, 1942, 31, 257-271.
29. WHITING, J. W. M. *Becoming a Kwoma; teaching and learning in a New Guinea tribe*. New Haven: Yale Univ. Press, 1941.

GLOSSARY OF SYMBOLS

Note: The literal signs are arranged primarily in the alphabetical order of the major letter constituting the sign, and secondarily according to the subscripts. The non-literal signs are grouped at the end of the list.

A = amplitude, magnitude, or intensity of a reaction; $A = k' s \ddot{E}_R - i'$.

a = empirical exponential constant in the equation expressing the generalized or effective habit strength as a function of sH_R and d , i.e.,

$$s\ddot{H}_R = sH_R e^{-ad}.$$

a' = empirical constant in the equation, $s\ddot{t}_R = \frac{a'}{s\ddot{E}_R b'}$.

B = empirical constant in the equation, $\dot{I}_R = \frac{cn}{B - W}$.

b' = empirical constant in the equation, $s\ddot{t}_R = \frac{a'}{s\ddot{E}_R b'}$.

iC_r = temporal coincidence of a receptor impulse (s) and the beginning of a reaction impulse (r).

c = empirical constant in the equation, $\dot{I}_R = \frac{cn}{B - W}$.

C_D = conditions which produce the drive (D), the objective conditions from which D may be calculated.

D = strength of dominant primary drive operative in the primary motivation to action after the formation of the habit involved.

D' = strength of primary drive (D) operative during the formation of a habit.

\dot{D} = the joint strength of all the non-dominant drives active at a given moment.

$$\overline{D} = 100 \frac{\dot{D} + D}{\dot{D} + M_D}$$

d = the number of j.n.d.'s lying between the two stimulus aggregates \dot{S} and S .

$$d' = \dot{D} - D.$$

e = a mathematical constant properly having a value of 2.7183 but here frequently given the value of 10 because more convenient in use where logarithms are involved.

sE_R = excitatory potential, potentiality of reaction evocation; i.e.

$$sE_R = s_1 + s_D \bar{H}_R \times \frac{\dot{D} + D}{\dot{D} + M_D}$$

$s\bar{E}_R$ = effective reaction potential, i.e., $s\bar{E}_R = sE_R - \dot{I}_R$.

$s\bar{E}_R^*$ = momentary effective reaction potential; $s\bar{E}_R$ as modified by sO_R .

F = the constant factor of reduction of the unrealized potential habit strength under given learning conditions. Thus if at each reinforcement the unrealized potentiality of habit strength is reduced by 1/10, F has a value of .1.

F' = amount of force.

f = an unstated quantitative functional relationship, e.g.,

$A = .141 sH_R + 3.1$ may be written, $A = f(sH_R)$, i.e., A is a function of sH_R .

f' = empirical constant in the equation, $n = c' s\bar{E}_R - f'$.

G = a need reduction or a stimulus which has been closely associated with a need reduction; primary reinforcement; also a primary goal reaction.

g = fractional portion of a goal reaction which may be split off from G and carried forward in a behavior sequence as a fractional antecedating goal reaction.

sH_R = habit strength conceived as a rough or approximate stimulus-response relationship to sH_r .

$s^N H_R$ = the habit strength (sH_R) which results from N reinforcements.

sH_r = habit strength conceived as a precise dynamic relationship between afferent and efferent neural impulses.

ΔsH_R = increment of habit strength resulting from a single reinforcement.

$s\bar{H}_R$ = effective habit strength: $s\bar{H}_R = sH_R e^{-f'd}$.

$ssH_R = s_1 + s_2 H_R$, i.e., the result of the summation of the habit strengths associated with two or more stimulus elements or

h = number of hours, as of food privation.

h' = empirical constant in the equation, $A = h' s\bar{E}_R - i'$.

I_R = amount of reaction inhibition.

\dot{I}_R = total amount of inhibitory potential, i.e., $\dot{I}_R = I_R + sI_R$.

sI_R = amount of conditioned inhibitory potential.

${}^{''''}I_R$ = amount of reactive inhibition t units of time after a given sequence of reaction evocations, i.e.,

$${}^{''''}I_R = I_R \times 10^{-at''''}.$$

ΔI_R = the increment of reactive inhibition generated at a single reaction evocation.

i = the exponential constant in a learning situation where ${}_sH_R = m - me^{-it}$. The quantitative value of i is given by the equation,

$$i = \log \frac{1}{1 - F}.$$

i' = empirical constant in the equation, $A = h' {}_s\bar{E}_R - i'$.

j = empirical constant in the equation expressing the maximum habit strength as limited by the delay in reinforcement (t):

$$m' = M'e^{-jt}.$$

j' = empirical constant in the equation expressing the stimulus generalization of habit strength,

$${}_s\bar{H}_R = {}_sH_R e^{-j'd}.$$

j.n.d. = discrimination threshold—the distance on a generalization continuum between two stimuli which, at the limit of practice, can be reacted to differentially on 75 per cent of the trials.

k = empirical constant in the equation expressing the maximum habit strength as limited by the quality and quantity of the reinforcing agent employed per reinforcement, i.e.,

$$M' = M(1 - e^{-kw}).$$

L = distance (length) of movement.

${}_sL_R$ = reaction threshold, the minimal amount of effective reaction potential (${}_s\bar{E}_R$) (the effect of oscillation being at a minimum) that will mediate reaction evocation.

log = logarithm.

M = the physiological maximum of habit strength attainable under optimal conditions. This value is taken as 100 habs.

M' = the maximum of habit strength with unlimited practice as limited by the amount and quality of the reinforcing agent, i.e.,

$$M' = M(1 - 10^{-kw}).$$

M_D = the physiological maximum of drive (100 mots).

M_I = the physiological maximum of reactive inhibition (100 pavs).

m = the physiological maximum of habit strength, attainable with unlimited practice, as limited by the asynchronism of \dot{S} and R in the reinforcement situation, e.g.,

$$m = m'e^{-\alpha t'} \quad \text{or} \quad m = m'e^{-\alpha''}.$$

m' = the maximum of habit strength, with unlimited practice, as limited by the delay in reinforcement, i.e.,

$$m' = M'e^{-h\tau}.$$

N = the number of reinforcements.

n = the number of unreinforced reactions required to produce experimental extinction, i.e.,

$$n = \frac{(B - W)({}_sE_R - {}_sL_R)}{c}.$$

O = a function of ${}_sO_R$ such that $O = {}_sO_R$ when ${}_sO_R < {}_s\bar{H}_R$, but
 $O = {}_s\bar{H}_R$ when ${}_sO_R > {}_s\bar{H}_R$.

${}_sO_R$ = the oscillatory weakening potentiality associated with effective reaction potential (${}_sE_R$).

P = coefficient of observable patterning, i.e., $P = Q - \bar{Q}$.

P' = theoretical index of patterning, i.e., $P' = 100 \left(1 - \frac{\bar{Q}'}{Q'}\right)$.

p = probability of reaction evocation.

Q = per cent of empirical reaction evocations by the positive or reinforced phase of a stimulus compound.

\bar{Q} = per cent of empirical reaction evocations by the negative or non-reinforced phase of a stimulus compound.

Q' = theoretical effective reaction potential of the negative portion of a stimulus patterning situation.

\bar{Q}' = theoretical effective reaction potential of the positive portion of a stimulus patterning situation.

q = empirical exponential constant in the equation of the dissipation of I_R , $I_R = I_R \times 10^{-qt''}$.

R = (1) reaction or response in general (muscular, glandular, electrical);
 (2) more specifically, the reaction which occurs as the result of previous conditioning.

\dot{R} = a reaction which is in the process of being conditioned to a stimulus.

R_* = unconditioned response such as the flow of saliva in a Pavlovian conditioned reflex experiment.

R_c = response to a conditioned stimulus before conditioning begins.

Ⓔ = a response which is either unobservable or exceedingly feeble and difficult of observation.

S = 1. stimulus energy in general, e.g., the energy of sound, light, or heat waves, pressure, etc.
 2. more specifically, stimulus energy which evokes a response on the basis of a previously formed habit.

S_A = stimulation arising from apparatus employed in an experiment.

S_D = drive stimulus, i.e., stimulation arising from a condition of need or disequilibrium.

S_c = conditioned stimulus such as the buzzer in a Pavlovian conditioned-reflex experiment.

S_u = unconditioned stimulus such as the food of the Pavlovian conditioned-reflex experiment.

\dot{S} = a stimulus when considered as in the process of being conditioned to a reaction.

s = afferent neural impulse resulting from the action of a stimulus energy on a receptor, as $S \rightarrow s$.

s_D = drive stimulus receptor discharge.

\dot{s}_c = afferent impulse arising from the action of a conditioned stimulus on a receptor.

s_A = afferent impulse arising from the action on a receptor of a stimulus energy arising from an apparatus employed in a learning situation.

\dot{s} = an afferent neural impulse when considered as in the process of being conditioned to a reaction.

\check{s} = an afferent neural impulse as modified by afferent neural interaction.

$\Delta(s \rightarrow r)$ or $\Delta(S \rightarrow R)$ = increment to a receptor-effector connection.

T = the time at which an instantaneous event occurs.

T_C = the time of the beginning of the R of a $\check{s}C$.

T_R = the time of the beginning of a reaction.

$T_{\dot{s}}$ = the time of the beginning of a stimulus which is in the process of being conditioned.

T_G = the time at which a reinforcement occurs.

t = (1) time in the sense of duration;

(2) the duration of the delay in reinforcement, i.e., $t = T_C - T_G$.

$t' = T_R - T_{\dot{S}} - .66$, when \dot{S} acts continuously and overlaps the beginning of R , where T_R and $T_{\dot{S}}$ are given in seconds.

$t'' = T_R - T_{\dot{S}} - .44$, when \dot{S} and R are practically instantaneous, where T_R and $T_{\dot{S}}$ are given in seconds.

t''' = the duration in minutes following a sequence of unreinforced evocations of R during which neither reinforced nor unreinforced evocations of R have occurred.

s^t_R = the latency of a reaction evocation, the time intervening between the beginning of the stimulus and the beginning of the response.

sU_R = unlearned or native receptor-effector reaction potential.

$s\dot{U}_R$ = momentary unlearned reaction potential.

u = empirical exponential constant in the equation expressing the maximum habit strength as limited by the time a stimulus (\dot{S}) has been continuously acting when R occurs, i.e.,

$$m = m'e^{-u\dot{S}}.$$

v = empirical exponential constant in the equation expressing the maximum habit strength attainable with unlimited reinforcement as limited by the degree of $\dot{S}-R$ asynchronism, i.e.,

$$m = m'e^{-v''}.$$

W = the amount of work, i.e., $W = F'L$.

w = the magnitude of a reinforcing agent employed as in the equation,

$$M' = M(1 - e^{-kw}).$$

Δ = increment, e.g., $\Delta_s H_R$.

ϵ = used in mathematical logic and read as "is," e.g., $x\epsilon S$ is read, "x is S."

Σ = the sum of a series, as $\Sigma \Delta_s H_R$, which means the sum of the increments of a habit strength resulting from a series of reinforcements.

σ_{sO_R} = the standard deviation of the oscillation of reaction potential.

$\phi(t)$ = the standard probability function.

$$\psi\sigma = \int_{-\infty}^{\sigma} \phi(t)dt.$$

\cdot = a sign used in mathematical logic meaning "and."

\supset = a sign of implication used in mathematical logic and read, "if . . . , then," ; e.g., $x \supset y$ is read, "If x, then y," i.e., x implies y.

\exists = a sign used in mathematical logic, e.g., $(\exists x)$, and read, "There is an x such that"

$\dot{+}$ = physiological summation, e.g., $sH_{R_1} \dot{+} sH_{R_2} =$

$$sH_{R_1} + sH_{R_2} - \frac{sH_{R_1} \times sH_{R_2}}{100}.$$

$>$ = greater than, e.g., $5 > 4$.

$<$ = less than, e.g., $4 < 5$.

\rightarrow = a causal receptor-effector relationship inherited or at least in functional condition at the outset of a learning situation.

$--\rightarrow$ = a causal receptor-effector relationship which is acquired by the organism.

\rightsquigarrow = a causal relationship other than that of a receptor-effector connection.

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